DISSERTATION ON
"ENDOSCOPIC REPAIR OF CSF RHINORRHOEA – A REVIEW OF ITS EFFICACY AND SUCCESS RATE"

Submitted in partial fulfillment of the requirements for

M.S. DEGREE BRANCH-IV OTORHINOLARYNGOLOGY

of

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

UPGRADED INTITUTUE OF OTORHINOLARYNGOLOGY
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MARCH- 2016

DECLARATION

I solemnly declare that the dissertation **"ENDOSCOPIC REPAIR OF CSF RHINORRHOEA – A REVIEW OF ITS EFFICACY AND ITS SUCCESS RATE"** is done by me at the Madras Medical College and Government General Hospital, Chennai during 2013- 2015 under the guidance and supervision of **Prof. Dr. M.K.RAJASEKAR M.S., D.L.O.**

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, towards partial fulfillment of regulation for the award of M.S. DEGREE IN OTORHINOLARYNGOLOGY (BRANCH-IV)

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ACKNOWLEDGEMENT

I am immensely grateful to **Prof.Dr.R.MUTHUKUMAR M.S. D.L.O. D.N.B.,** The Director and Professor, Upgraded Institute of Otorhinolaryngology, for his valuable guidance, suggestions , encouragement and help in conducting the study.

I am greatly indebted to **Prof. Dr. M.K.RAJASEKAR M.S. D.L.O.,** Professor, Upgraded Institute of Otorhinolaryngology, for the encouragement and support all through my study under his guidance and support during the conduction of the study.

I express my sincere gratitude to Ex-Director and Professor **Dr.G.GANANATHAN M.S. D.L.O.** for his valuable support in conduction of the study.

I would like to express my sincere gratitude to **Prof.Dr.VIMALA M.D.,** the DEAN, Madras Medical College , for having permitted me to use the hospital material in this study.

I express my sincere thanks to all the Assistant Professors, for their thoughtful guidance throughout the work.

I thank the Secretary and Chairman of Institutional Ethical Committee, Government General Hospital and Madras Medical College, Chennai.

I thank all my colleagues and friends for their constant encouragement and valuable criticism.

All I am is because of my parents who were always besides me, at times of good and bad. I take immense pleasure to thank my mom and dad for their unconditional love, care, support, guidance and valuable piece of advice that has led to the fulfillment of this study.

I thank my sister, brother, husband and all my relatives who encouraged and helped me through this study.

I express my gratitude for the generosity shown by all the patients who participated in the study.

I am extremely thankful to my family members for their continuous support. Above all I thank God Almighty for his immense blessings.

CERTIFICATE

This is to certify that this dissertation entailed **"ENDOSCOPIC REPAIR OF CSF RHINORRHOEA – A REVIEW OF ITS EFFICACY AND SUCCESS RATE"** submitted by **Dr. HEMALATHA K,** appearing for M.S. ENT., Branch IV Degree examination in March 2016 is a bonafide record of work done by her under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University , Chennai , Tamil Nadu, India.

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ABSTRACT

"ENDOSCOPIC REPAIR OF CSF RHINORRHOEA – A REVIEW OF ITS EFFICACY AND SUCCESS RATE"

BACKGROUND

CSF rhinorrhoea occurs due to a trans dural communication between the nasal cavity and the subarachnoid space. Its repair has been revolutionized by the advent of much easier endoscopic repair techniques which cause lesser morbidity as compared to open surgical techniques with regard to the CSF leak sites under study. This study strives to elucidate the outcomes obtained through endoscopic approach and also our experience obtained ,during the management , through our patients under study.

INTRODUCTION

Duramater of the brain is an important barrier for any infection to ascend intracranially. It contains the cerebrospinal fluid present within the subarachnoid space and hence, a transdural event can cause breach in its integrity causing leak of the cerebrospinal fluid, as evident from watery nasal discharge.

CSF rhinorrhoea ensues when the breach involves the nasal mucosa , periosteum, bone forming the skull base in the region of nose and paranasal sinuses , endosteum, duramater and arachnoidmater.

Transdural event can be due to trauma, nasal surgery, tumors invading skull base or may be spontaneous. In each of these cases, the outcome is the same- CSF leak from the site of injury to dura and ascending infection through the defect causing meningitis and related complications. In cases where the medical therapy fails, it is mandatory for surgical closure to prevent such complications.

Surgical repair of CSF leak site can be either through an intracranial or an extracranial approach. Endoscopic approach is a type of extracranial approach which has the advantage of being

less invasive , no external surgical scar , excellent site localization with preservation of the surrounding anatomy and shorter hospital stay. Unlike open surgical techniques, endoscopic approach avoids excessive mobilization of the brain and the dura and, offers wide and site specific view through a smaller exposure than that achieved through a microscope.

AIM OF THE STUDY

1) To study the common sites of CSF leak

2) To evaluate the efficacy of endoscopic CSF leak repair.

3) To study various methods of skull base defect closure used in endoscopic repair of CSF rhinorrhoea.

INCLUSION CRITERIA

1) Anterior and middle cranial fossa easily approached endoscopically

2) Size of defect- small size defect as determined using direct endoscopic visualization and radiological evaluation.

3) Etiology- traumatic, iatrogenic, spontaneous and tumor-related CSF leaks.

4) Precise defect that can be localized.

5) Failure of conservative management.

EXCLUSION CRITERIA

1) Defect not localized by radiological and other CSF leak studies.

2) Multiple injuries requiring intracranial approach

3) Pneumocephalous

REVIEW OF THE LITERATURE

CSF rhinorrhoea was first described in the second century AD by Galen. His theory was that , the CSF leaked into the nose through the pituitary and ethmoid regions.

1826 -CSF rhinorrhoea in a child with hydrocephalous was first described by Charles Miller, who presented with an intermittent discharge of nasal fluid. Autopsy revealed communications between the nasal and cranial cavities.

In 1899 , St. Clair Thompson introduced the term ‘rhinorrhoea’ and the first series of patients with spontaneous CSF leaks . Differentiation between cerebrospinal rhinorrhoea and nasal rhinorrhoea was made by him.. He did not recommend surgical intervention.

First intracranial repair by bifrontal craniotomy was done by Walter Dandy in 1926. It was the procedure of choice till 1940, but morbidity and incidence of anosmia was high..It had a recurrence rate of 27%, and success rate of 12 to 20%

1948 - Gusta Dohlman used nasoorbital incision for external ethmoidectomy and was the first to describe the extracranial approach.

1952 - Transnasal approach was used by Oscar Hirsch for acromegalic patient with two sphenoidal leak.

1981- Wigand and Stankiewicz closed endoscopically the minor CSF leaks that occurred during ethmoidectomy. Wigand used fibrin glue to close the leak.

1926- The first successful intradural closure was done by Dandy by suturing autologous fascia lata behind the posterior wall of the frontal sinus.

The use of endoscopic telescope in trans septal and trans sphenoidal surgery to localize sphenoid CSF leakage was reported by Papay et al . The combination of intrathecal fluorescein and nasal endoscopes was introduced by . Messerklinger, Reck and Wissen-Siegert , to diagnose anterior cranial fossa CSF leak.

In 1937 Cairns, a British neurosurgeon, divided CSF leak into acute traumatic, postoperative, delayed traumatic, and spontaneous. And hence, was the first to provide a classification for the same.He also reported the extradural placement of fascia lata

for CSF repair. Ommaya and then Vrabec and Hallberg later modified this classification. In his series, Ommaya reported a 2% incidence of CSF leaks in all head injuries. When involving only patients with skull base fractures, this incidence rose to 5%.He also noted that 90% of CSF leaks were due to trauma.

In 1944, Schroeder described a patient with CSF rhinorrhea who recovered after treatment with sulfonamides and a lumbar puncture. Also in 1944 Dandy reported that the surgical repair of any CSF leak within 2 weeks of its onset can prevent meningitis.

Endoscopic method of closure was reported in detail by Mattox and Kennedy through their study, in which they described the management of encephalocele and techniques to seal the leak.

ANATOMY OF ANTERIOR SKULL BASE AND PARANASAL SINUSES

OVERVIEW

The anatomical anterior skull base is bounded anteriorly by hard palate and the alveolar process while a surgical anterior skull base consists of that part formed by the cribriform plate of ethmoid bone and the orbital part of frontal bone. From the anterior surface of sphenoid bone starts the lateral skull base. Anterior skull base is an approach to frontal sinus, cribriformplate, frontal lobe, ethmoids and sphenoid sinus concerned with CSF leak repair.

EMBYOLOGY

The facial development starts at around 4-8 weeks of intrauterine life. The frontal bone develops from an intramembranous ossification while, the ethmoid and the sphenoid develops from an enchondral ossification.

Paranasal sinus and the turbinates development from lateral wall ridges called the ethmoturbinals ,which are formed during the 9th to 10th week of gestation. Five to six ridges appear during the development, and through regression and fusion only 3-4 persist. The respective furrows associated with these turbinals extend to form the recesses and the paranasal sinuses.

During the 13th week, the descending part of the first ethmoidal furrow becomes ethmoidal infundibulum. Its superior ascending part becomes the frontal recess. Further pneumatization of the frontal recess into the frontal bone finally results in the formation of frontal sinus. The frontal sinus is formed as a small blind sac within the frontal bone till the child is about 2 years of age, then secondary pneumatization begins and proceeds until 9 yrs.

Additional furrows and corresponding ridges between them evolve into anterior ethmoidal and infundibular cells.

In addition, a maxilloturbinal arising below the ethmoturbinals form the inferior turbinate. Sphenoid sinus developes at around fifth year of life

OSTEOLOGY

THE ETHMOID BONE- It is a delicate bone consisting of three parts – the ethmoidal labyrinth, cribriform plate and the perpendicular plate. The ossification centers arise one for perpendicular plate at 2 months of age and one for each labyrinth. The cribriform plate ossifies partly from the ossification centre of the perpendicular plate and partly from that of labyrinth and proceeds from lateral to medial and, from anterior to posterior.

During development and until the first year of age, the ethmoid bone consisted of two separate symmetrical halves. At the end of the first year both halves of the ethmoid bone unite with the formation of the crista galli, lamina mediana and complete ossification of the cribriform plate.

Cribiform Plate (*lamina cribrosa; horizontal lamina*).—It is the horizontal part forming the roof of nasal cavity which anteriorly fits into the ethmoidal notch of frontal bone. Projecting upward from the middle line of this plate is a thick, smooth, triangular process, the crista galli**,** so called from its resemblance to a cock's comb, for the attachment of the falx cerebri. Its two small projecting alae are received into corresponding depressions in the frontal bone and complete the foramen cecum.

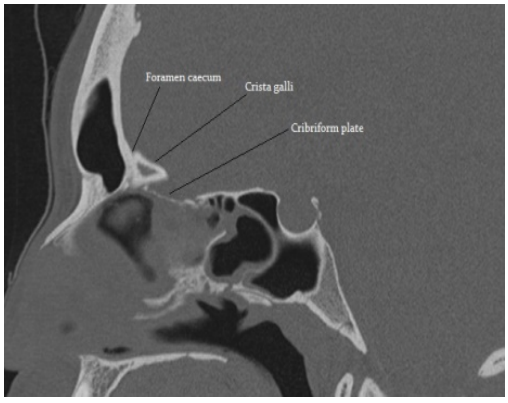


Fig1: coronal section of CT PNS

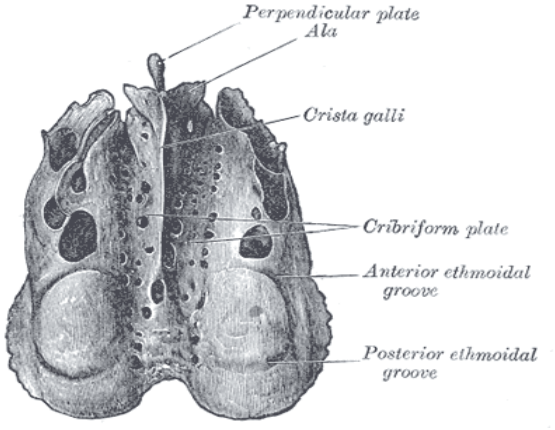


Fig2: Base of skull in the region of cribriform plate

On either side of the crista galli, it supports the olfactory bulb and is perforated by foramina for the passage of the olfactory nerves. The foramina in the middle of the groove are small and transmit the nerves to the roof of the nasal cavity; those at the medial and lateral parts of the groove are larger—the former transmit the nerves to the upper part of the nasal septum, the latter those to the superior nasal concha.

The labyrinth has an upper surface which articulates with the ethmoidal notch of the frontal bone and completing the canals for the anterior and the posterior ethmoidal vessels at the frontoethmoid suture line. The posterior surface articulates with the sphenoidal concha and the orbital plate of palatine bone. The thin lateral plate, the lamina papyracea , forms part of medial wall of orbit. The ethmoidal cells are open cranially which are closed by the orbital plate of frontal bone forming the fovea ethmoidalis. The lateral roof of the dome of ethmoidal air cells is thus provided primarily by the frontal bone while its medial roof is formed by the lateral lamella of lamina cribrosa.

The **middle turbinate** and the superior tubinate are parts of the ethmoid bone.Of the three parts of the middlt turbinate, the anterior third is saggitally oriented and gets inserted into the skull base at the lateral edge of the lamina cribrosa. Excessive manipulation of this region will cause iatrogenic injury to the skull base and may lead to CSF leak.

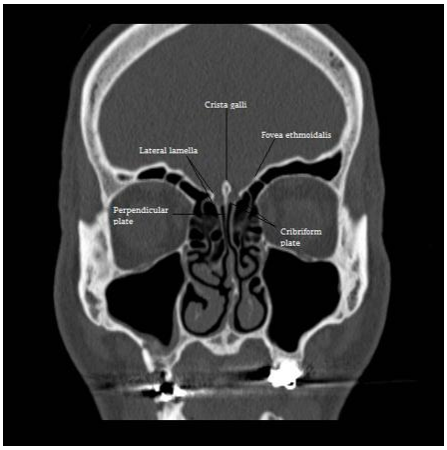


Fig3: showing the olfactory fossa

Kero's classification of olfactory fossa is based on the fact that, the horizontal portion of the lamina cribrosa is at a lower level, than its lateral lamella and the fovea ethmoidalis. The depth of olfactory fossa is determined by the height of lateral lamella. In type I , the olfactory fossa is 1-3mm deep; type II is 4-7 mm ; type III- 8-16mm.

Hence, iatrogenic injury is more common in case of deep olfactory fossa , Kero's type III , and particularly at the thinner

lateral lamella.It is estimated that the average thickness of frontal bone forming the roof of ethmoidal air cells is 0.5mm; lateral lamella is 0.2mm and the ethmoidal sulcus which lodges the anterior ethmoidal artery and the most vulnerable part of anterior skull base is 0.05mm thin.

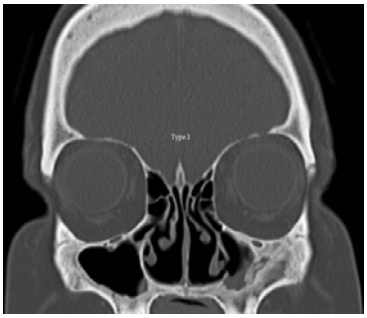


Fig4:Kero's type I

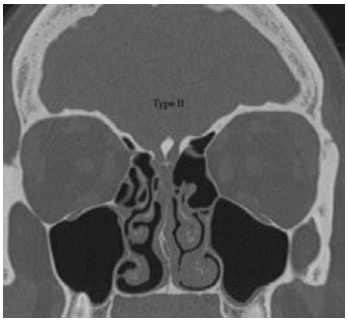


Fig5:Type II

Fig6: Type III

During its course, the anterior ethmoidal artery crosses three cavities (the orbit, ethmoidal labyrinth and anterior cranial fossa of skull.It is a branch of the ophthalmic artery , given off in the orbit and enters the nasal cavit through the anterior ethmoidal foramen present at the frontoethmoidal suture line .It passes within a bony mesentry (orbitocranial canal) along the roof of nasal cavity connecting the anterior ethmoidal artery to the skull base with a space of about 5 mm between the the two. In CT scan, the exit of the artery from the orbit is evident as the Kennedy's nipple, a bony projection at the junction of medial rectus and superior oblique muscle. It enters the olfactory fossa through the lateral lamella of

cribriform plate and runs forward in a groove called the ethmoidal sulcus, giving off the anterior meningeal artery. It again enters the nasal cavity through the cribroethmoidal foramen and the lamina cribrosa.

The relationship of the anterior ethmoidal artery to the roof of the ethmoid is highly variable and is at risk during endoscopic sinus surgeries. Identification of this artery is important in identifying frontal sinus outflow tract and superior limits of skull base and in avoiding the risk of bleeding and orbital hematoma.

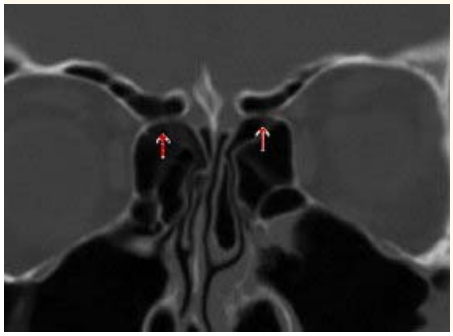


Fig7:showing course of anterior ethmoidal artery (arrow) and Kennedy's nipple.

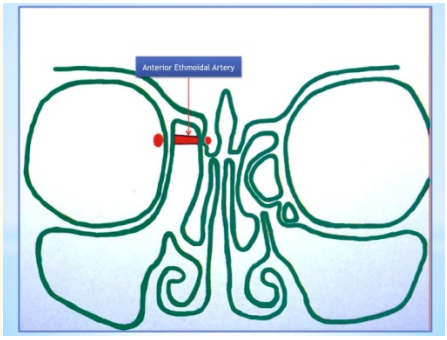


Fig8: Course of anterior ethmoidal artery

The region of cribriform plate ,superior attachment of middle turbinate, the fovea ethmoidalis and the anterior ethmoidal artery is important in the context of CSF leak particularly of iatrogenic etiology. Also, in blunt trauma , fractures commonly occur at the transition from a thick bony segment of fovea ethmoidalis to a

much thinner lateral lamella. The dura is much thinner and strongly attached in this area and the anterior ethmoidal artery can be torn where it enters and leaves the olfactory fossa.

THE FRONTAL BONE

It ossifies by two primary centers, one for each half, at 2 months of intrauterine life. They are separated by frontal suture which later unite by 8 years .It consists of three parts- squamous, orbital and the nasal parts. Our main consideration are the orbital part, the frontal recess and the frontal sinus. The frontal sinus is absent at birth and is formed with the development of anterior ethmoid cells after the age of 2. Growth of this sinus increases at the age of 6 and continues until the late teenage years

Endoscopic approach to the skull base needs more precise understanding of its anatomy so as to approach the site of CSF leak, as well as to avoid complications such as anterior ethmoidal arterial bleed and secondary frontal sinusitis post-surgery. The frontal sinus drainage pathway and its variations is largely based upon the anatomic variations of the cells around the frontal recess.

Frontal sinus drains into the ‘hour-glass’ shaped frontal recess formed from the remnant of ascending portion of first

interturbinal furrow. Frontal recess tapers as it reaches the frontal ostium, which is the narrowest part, and again widens as the anterior and the posterior tables diverge . Frontal recess is bounded laterally by lamina papyracea, medially by the lateral surface of anterior part of middle tubinate, anteriorly by the posterosuperior wall of agger nasi and posteriorly by anterior wall of bulla ethmoidalis.If the bulla lamella doesnot reach the roof, it communicates with the suprabullar recess.

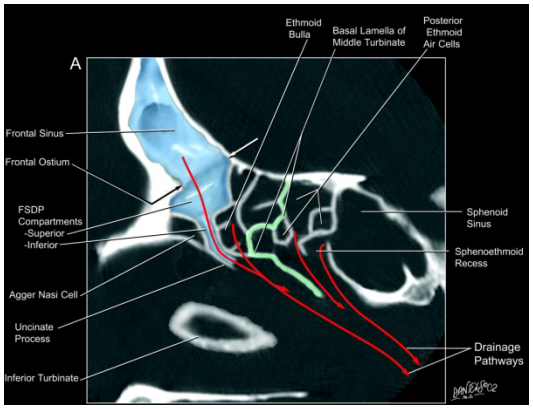


Fig9:Frontal sinus drainage pathway

The frontal sinus drainage pathway (FSDP) has superior and inferior compartments. The *superior compartment* of the FSDP is formed by the union of adjacent air spaces at the anteroinferior portion of the frontal bone and the anterosuperior portion of the ethmoid bone. Its upper border is the frontal ostium When the anterior portion of the uncinate process extends superiorly to attach

to the skull base or the middle turbinate, the inferior compartment of the FSDP is the *ethmoid infundibulum*. This then communicates with the middle meatus via the hiatus semilunaris;.However, when the anterior portion of the uncinate process is attached to the lamina papyracea , the inferior compartment of the FSDP is the middle meatus and the ethmoidal infundibulum ends as terminal recess

The shape of frontal recess is largely determined by the pneumatization of the agger nasi, bulla ethmoidalis and ethmoidal air cells around the frontal recess called the 'frontal cells'.Such anatomical variations can narrow down the frontal recess into a tubular lumen called the 'nasofrontal duct'.Extensively pneumatized agger nasi can be large enough to be mistaken for frontal sinus itself, and in its incomplete removal, the residual posterosuperior wall of the agger nasi cell scar posteriorly to ethmoidal cell and cause iatrogenic frontal sinusitis.

The anatomic situation is further complicated by the anterior ethmoidal air cells which develop from the frontal recess. Stammberger sates that "cells develop into the frontal bone, alongside the fontal sinus. These were called 'the bulla frontalis' by Zuckerkandl".Skull base always forms the roof of these cells and

fracturing of its walls posteriorly must be done gently to avoid injury onto the skull base.

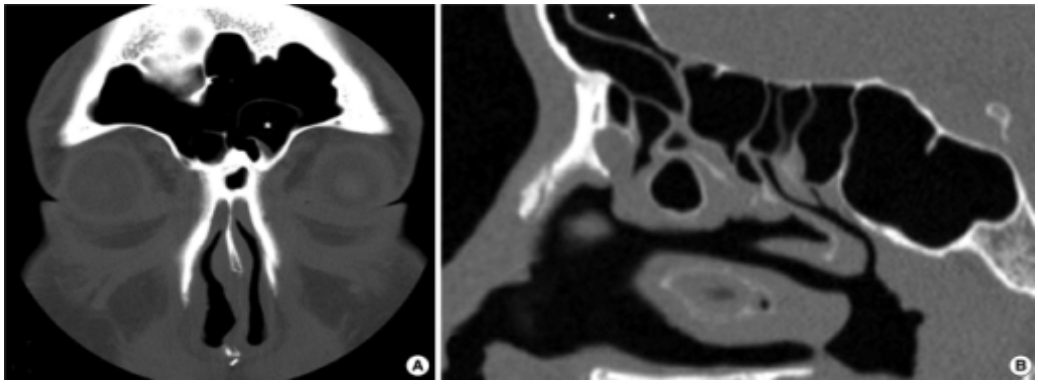


Fig 11 A,B: Posteriorly based frontal bulla cell in coronal and saggital sections.

Frontal bulla cell must not be confused with anteriorly based type 4 frontal ethmoidal cell which pneumatizes more than 50% of the height of the frontal sinus. In some cases it is difficult to differentiate frontal sinus from bulla frontalis.

Peter-John Wormald insist on meticulous dissection in the region of frontal recess and that, the instrument should not be placed through the roof of the cell as it may enter the anterior cranial fossa if there is no space between the roof of the cell and the skull base . Hence, careful dissection should be done assessing the superior attachment of the septa of each cell.

SPHENOID BONE

The sphenoid bone is an unpaired bone present in the midline composed of a body and greater wing, lesser wing and pterygoid plates on each side. The sphenoid sinus develops within the body of sphenoid bone at the limit between the anterior and middle cranial fossa. It is present at birth and arises from recess between the sphenoid concha and the presphenoid body and reaches its full size by the late teenage year.

The sphenoid sinus is divided by the intersphenoid septum with a wide range of variations. It may extend obliquely and get inserted onto the bony canal for internal carotid artery or the optic nerve. Hence, manipulation of the intersphenoid septum must be done carefully to avoid complications. It may have minor incomplete septations also.

The lateral wall of sphenoid sinus is related to the optic nerve, cavernous sinus with its contents- the internal carotid artery, the third , fourth and the sixth cranial nerves. The optic canal and the internal carotid artery produce a visible bulge in the lateral wall. There is an estimated dehiscence in the bony canal wall of internal carotid artery upto 25% and that of the optic nerve of around 6%. Hence, manipulation in these areas must be done cautiously to avoid any compications.

Pneumatization of the sphenoid sinus can extend further from its body, and into all of its parts, such as the clinoid processes, greater wings and pterygoid plates. When it extends laterally between the foramen rotundum and the vidian canal, onto the pterygoid plates , lateral recess is formed which may also extend posterior to the maxillary sinus.The posterior ethmoidal cell can pneumatize along the lateral wall of the sphenoid sinus forming the ‘onodi cell’.

Fig 12:Onodi cell


Fig 13:Lateral recess in a female patient With spontaneous CSF leak.

In 1888, Maxmillan Sternberg described the lateral craniopharyngeal canal as a congenital bony defect in the lateral wall of sphenoid sinus.He described it to extend from the junction of body of the sphenoid bone and the posterior root of the lesser wing , just medial to the superior orbital fissure, the foramen rotundum and the vidian canal . It was postulated for spontaneous CSF leak from the lateral wall of sphenoid sinus however, its existence is still debated as lateral recess is

often radiologically proven as the site of origin of the leak. The communication with the middle cranial fossa is the more likely if the sphenoid sinus is laterally pneumatized and is associated with arachnoid pits along its anteromedial aspect.

PHYSIOLOGY OF CEREBRO SPINAL FLUID

Cerebrospinal fluid is a clear and colourless fluid present in the ventricles and cisterns within the cranial cavity and the subarachnoid space around the brain and the spinal cord. Its main function is to cushion the brain and act as shock absorber .It occupies a total of 90-150 ml of the entire cerebral cavity enclosing brain and the spinal cord with a daily production of 450-550 ml at a rate of approximately 0.3-0.4 ml/hr.

Two-third of the total production is from the choroid plexus in the ventricles. The remaining CSF is produced by the ependymal layer, the arachnoid membrane and brain itself from the perivascular spaces. The secretion is by active transport of Na^+ ions across the epithelial lining cells of the choroid plexus. The Na^+/K^+ATPase at the apical membrane actively transports the Na^+ ions out from the cell into the ventricles and the water follows this gradient. Carbonic anhydrase within the cell catalyses the formation of HCO^{-3} ions required for the transport of water molecules across the cell. The production maintains a circadian rhythm with a maximal production at 2 am.

Diffusion either by passive or facilitated transport for the proteins, and the active transport for the glucose , helps in transfer of molecules during the production of CSF. This ultrafiltrate of the plasma forms the cerebrospinal fluid with a composition and properties as follows-

- pH- 7.33
- Osmolality- 289 mOsm/kg (similar to that of plasma)
- Pressure – 10 mmHg (130 mm of H_2O)
- Specific gravity – 1.007
- Glucose -40-60 mg/100ml ($2/3^{rd}$ of blood glucose value)
- Protein -15-45mg/dl
- Na^+ -147 mEq/l
- K^+ -2.8 mEq/l
- Cl^- - 113 mEq/l
- HCO_3^- -23.3 mEql
- WBC –0-5/µl
- Culture- negative

The CSF flows from the lateral ventricles to the third ventricle, and through the aqueduct of Sylvius to the fourth ventricle . From there it reaches the cisterna magna, the largest of the subarachnoid cisterns and flows to the subarachnoid space from where it gets absorbed into the saggital sinus. During its course, the CSF circulation is explained by two different concepts – a unidirectional bulk flow circulation and a back and forth pulsatile circulation related to the cardiac cycle of the cerebral arteries.

Normally, the CSF absorption must match the production to maintain a constant CSF pressure. This is maintained by the valve-like action of the arachnoid villi . A pressure gradient of 1.5mmHg or more, between the arachnoid villi and the draining dural venous sinuses , will allow absorption of the CSF into the venous sinuses at a rate determined by the gradient of the pressure between the two. Any mismatch between the production and absorption of CSF might lead to increased intracranial pressure of 20 mmHg or more.

CSF pressure is a direct measure of the intracranial pressure, which determines the cerebral blood flow. Cerebral perfusion pressure is defined as the difference between the mean arterial pressure and the intracranial pressure.

ETIOLOGY OF CSF RHINORRHEA

Ommaya et al classified the etiology of CSF rhinorrhoea into traumatic and non-traumatic which he sub-classified :

TRAUMATIC

- a. Accidental
  - acute
  - delayed
- Iatrogenic
  - Acute
  - Delayed

NON-TRAUMATIC

- High pressure
  - Tumors
  - Direct
  - Indirect
  - Hydrocephalous
- Normal pressure

- Congenital anomalies
- Focal atrophy
- Olfactory
- Sellar
- Osteomyelitic erosion
- Idiopathic

It can also be classified [6] as,

CONGENITAL

meningocoele, meningomyelocoele,congenital skull base efect, congenital hydrocephalus

ACQUIRED

- IDIOPATHIC- cause unknown
- TRAUMATIC

Surgical cause- endoscopic sinus surgery, transcranial approaches

Non-surgical causes- skull base fractures pen or penetrating injuries

Post-traumatic hydrocephalous

Inflammatory- erosive lesions- mucoceles, polyp, cystic fibrosis, fungal sinusitis, osteomyelitis post-infective hydrocephalus

Neoplasm- invading skull base causing hydrocephalus

TRAUMATIC LEAK

Accidental Traumatic causes constitutes of about 96% of all cases ,of which about 80% of all traumatic CSF rhinorrhoea belong to the accidental etiology, penetrating and closed-head trauma cases. Due to the adherence of dura to the bone in the region of anterior skull base, fracture in this region often result in dural tear , and hence, a communication between the subarachnoid space and the sinuses resulting in CSF rhinorrhoea. Most of them occur in the region of fovea ethmoidalis followed by posterior wall of the frontal sinus. Middle cranial fossa as well as posterior cranial fossa fractures involving the clivus can cause CSF rhinorrhoea via the sphenoidal sinus commonly via its lateral extensions.

In such cases, CSF rhinorrhoea is often immediate (<48 hrs) and usually resolves with conservative management. It has resolution rate of 50% – 70% within one wee,k and 90%-95% within 6 months. Of the delayed type 95% usually presents within

3 months, and it is due to the resorption of the bone and soft tissues around the site of fracture caused due to a decrease in blood supply. In those cases which doesnot resolve following medical therapy, brain herniation ,with or without a displaced fracture, is said to affect the healing of the dura.

Iatrogenic - Traumatic CSF rhinorrhoea of iatrogenic variety commonly occur following transphenoidal surgery , extended endoscopic sinus surgeries and related skull base surgeries, revision endoscopic sinus surgeries due to altered anatomical landmarks. This has now become an increasing cause and nearly 33% were following pituitary surgeries as reported by Lantz et al. About 50% of the leaks presents within first week following the injury.

The common site following neurosurgical procedure is the sella tursica, while that of endoscopic sinus surgeries are the lateral lamella of cribriform plate and fovea ethmoidalis[12]. In cases of skull base defect caused iatrogenically, it is adviced to close the defect intraoperatively to prevent CSF rhinorrhoea as well as complications such as pneumocephalous , as spontaneous healing of the defect is far less as compared to the accidental type.

NON-TRAUMATIC / SPONTANEOUS LEAK-

It was earlier considered to occur without any known cause, and hence idiopathic. Theories for primary non-traumatic CSF leaks include focal atrophy, rupture of arachnoid projections that surround the olfactory nerve fibres in the cribriform area and the persistence of embryonic olfactory lumen. They constitute around 3-4 % of the total leaks .

It is now being postulated that, it can also be due to an elevated intracranial pressure .High intracranial pressure results in remodeling and thinning of the bones at the skull base , and can cause the leak which actually acts as a safety valve. An intracranial neoplasm, hydrocephalous or the benign intracranial hypertention act on the prolongation of arachnoid sleeves around the olfactory fibres. The dura at this area ruptures and lets out the CSF to decrease the intracranial pressure. Hence, this type is more common in the cribriform area and are intermittent presenting over months to years.

Patients with hyperpnuematization of the paranasal sinuses have an increased risk of spontaneous CSF leak, particularly in the region of lateral recess of sphenoidal sinus. Ultimately, the weakened bone leads to bony defect, onto which the dura herniates (meningocele) until it tears, precipitated by sudden raise in

intracranial pressure while sneezing, coughing, straining etc.Such patients may also develop herniation of the brain parenchyma (encephalocele) in to the sinuses which interfers any spontaneous healing of the dura . Hence, spontaneous resolution of these leaks are less as compared to the traumatic variety due either an increased ICP or the encephalocele.

Normal pressure leaks occur at region of a congenital defect , commonly in the region of cribriform plate and the sella turcica where the extension of subarachnoid space occurs around the pituitary stalk. They may present with or without a meningoencephalocele. Focal atrophy in the region of olfactory or the sella , theory as described by Ommaya in 1968. Spontaneous CSF leaks generally require surgical closure as its rate of spontaneous resolution is less than 33%.

OTHER CAUSES OF CSF RHINORRHOEA OF INTEREST

A.Empty sella syndrome(ESS)- The term empty sella was first applied to an anatomic finding at autopsy by Busch in 1951. Maiea et al reported CSF leak in 73% of patients with an empty sella. In patients with no known pituitary disease, the pituitary gland was severely flattened against the floor of the sella ; wide aperture at the diaphragma sella ,with an enlarged intrasellar

subarachoid space and arachnoid granulations. Progressive erosion due to the pulsation of arachnoid granulation can eventually lead to the CSF fistulae. It was regarded as an effect of raised intracranial pressure, while recent school of thought consider it to be of a primary ESS or a secondary ESS due to pituitary lesions or radiation, and it can be due to either high pressure or normal pressure mechanics . It is more commonly asymptomatic and occurs mostly in obese females in the 4^{th} decade. Less than a third of these patients develop symptoms related to raised ICP or pituitary insufficiency.

Radioloically, the sella may be enlarged symmetrically with an identifiable pituitary stalk traversing towards the residual pituitary. In MRI, it shows isointensity of the fluid with cerebrospinal fluid (T1-weighted hypointensity and T2-weighted hyperintensity)Such cases might require aggressive medical and surgical treatment such as placing a lumbar drain or shunting if required to reduce the incidence of recurrence in CSF leak closure.

B.Idiopathic intracranial hypertension(IIH) - is demographically more common in obese females. Diagnostic criteria for IIH was initially given by Walter Dandy in 1937 which

was later modified by J.L. Smith in 1985 , including the advanced radiological imaging. The patients satisfy all of the criteria below,

MODIFIED DANDY'S CRITERIA FOR IIH

1) Signs and symptoms of raised ICP – headaches, nausea, vomiting, visual changes, papilledema(occurs in nearly all patients).

2) Elevated CSF pressure on lumbar puncture- (>25cm H_2O opening pressure)

3) Normal CSF chemical and cytological composition

4) Absence of localizing or focal neurological signs (excludes sixth nerve palsy)

5) Exclusion of cerebral venous thrombosis on neuroimaging (requires CT /MRI).

Its is postulated due to reduced CSF absorption, increased CSF production or increased venous pressure due to stenosis of large cerebral venous sinuses.

TUMOR-RELATED CSF RHINORRHOEA

Highly erosive benign lesions of the nose and paranasal sinuses such as inverted papilloma, as well as malignant tumors in these region can invade the base of skull with bony erosion with extension into the anterior cranial fossa . The tumor can cause CSF rhinorrhoea if it has exended transdurally with communication of the subarachnoid space. Otherwise, even in extradural tumors , tight adhesions onto the dura can cause intraoperative CSF leak during dissection of the tumor away from the dura.

Fig 14: Transitional cell carcinoma of nase and paranasal sinus with erosion of cribriform plate and intracranial extension

There is a high risk of postoperative CSF rhinorrhoea , as well as pneumocephalous if the bony defect is not well sealed intraoperatively. With the advent of endoscopic CSF leak repair, there is an added advantage of endoscopic resection of tumors with

greater intracranial extension, without the need for an open transcranial approach for the same.

CONGENITAL CAUSES[16]

Congenital dehiscence in the skull base usually occurs in the region of anterior neuropore . They occur due to developmental malformation of the skull-base , which presents with a funnel-shaped defect through which the dura and the brain parenchyma herniate. Common locations include the cribriform plate in the region where the olfactory filaments pass along with the arachnoid sleeves at the region of foramen cecum. They may present with multiple areas of CSF leaks and with normal ICP except in cases with hydrocephalous.

ROUTE OF CSF RHINORRHOEA

CSF leak can occur from the respective cranial fossa as follows,

Anterior cranial fossa via

1) Frontal sinus
2) Cribriform plate
3) Roof of ethmoidal sinus
4) Sphenoid sinus

Middle cranial fossa via

1) Sphenoid sinus

2) Mastoid air cells, middle ear , Eustachian tube

Posterior cranial fossa via

1) Spenoid sinus

2) Mastoid air cells, middle ear , Eustachian tube

CSF leak through the mastoid air cell system reaches the middle ear, and drains through the eustachian tube into the nasopharynx. From the nasopharynx it drains through the nose and appears as rhinorrhoea. This study doesnot include CSF leak through the mastoid air cell system .

HISTORY TAKING[13]

A detailed history taking is necessary for diagnosis of CSF rhinorrhoea. Due to clear nasal discharge, it can be misdiagnosed as allergic rhinitis or vasomotor rhinitis. In CSF leak, the patient gives a history of clear watery nasal discharge, usually unilateral which is aggravated with bending forwards ,lifting weight ,staining or wetting of pillow on lying down. It cannot be sniffed back unlike the other causes of rhinorrhoea.

There can be antecedent history of trauma with or without nasal bleed , or a previous history of nasal surgery. Delayed type of CSF leak can occur after a few months to years of the accident and commonly lead to misdiagnosis if complete history is not elicited. Drainage may be intermittent as the CSF accumulates in one of the paranasal sinuses and drains externally on changing the position of the head (reservoir sign)

History of elevated intracranial pressure include the headache, visual disturbance such as blurring of vision, diplopia , loss of peripheral vision and vomiting. History of neurological deficits and post spinal headache may be an indicator of raised ICP. Neurological deficits in particular can lead to the localization of the lesion.

Lesion can be localized with the help of proper history. Olfactory region involvement due to lesion in the cribriform plate or the anterior cranial fossa presents with anosmia (in 60% of post-traumatic cases).Involvement of the optic nerve is seen in lesion involving the sella and the suprasellar area, sphenoid sinus, and the posterior ethmoid cells.

History of fever, projectile vomiting, altered sensorium indicates development of meningitis. Recurrent meningitis, especially pneumococcal[15] meningitis can be due to a direct communication between the nasal cavity and the intracranial space and has to be evaluated even if there is no CSF leak. Bernal et al[14] states that "neither the conservative approach nor the transcranial repair was able to prevent this considerable incidence of ascending meningitis. We believe that the high incidence of meningitis is not acceptable; thus, we are now evaluating early intervention using endoscopic techniques for the identification and/or repair of post-traumatic fistulas."

PHYSICAL EXAMINATION

It includes complete rhinological, otological, head and neck and neurological evaluation.

Eliciting the reservoir sign .This is done by asking the patient to rise from a lying position and flex the neck forwards. The CSF which gets accumulated within the paranasal sinuses starts to drip on the side of lesion .Patient with intermittent CSF rhinorrhoea may be unrevealing in the routine physical examination. Moreover, paradoxical rhinorrhoea can occur when midline structures are dislocated (eg. Crista gali, vomer).This causes CSF leak through

the other nostril. Bilateral CSF leak can also occur and gives no clue of laterality.

Fig 15: Reservoir sign

- Handkerchief test- Due to high content of mucin in cases of allergic and infective causes of rhinitis, dried nasal secretions over the handkerchief causes stiffening , while In CSF rhinorrhoea , since it contains only minimal mucin due to contamination of the nasal secretions, it doesnot stiffen the handkerchief.

- Halo sign- It is elicited in traumatic CSF leaks. The nasal discharge is made to drip over a filter paper. Blood due to trauma fills the centrals part , while the clear CSF forms a halo or ring around the blood as it spreads along the filter paper.

- Signs of meningitis- This is more common with traumatic CSF leak persisting more than 1 week due to ascending infection from the nose and paranasal sinuses. They include fever, headache, neck rigidity, irritability or altered sensorium, positive Kernig's and Brudzinski's sign.

LABORATORY INVESTIGATIONS[18,19]

Glucose determination-Glucose content of CSF fluid is determined by the glucose oxidase test .A concentration of 50-80 mg/dl is consistent with CSF rhinorrhoea. But, this method is not a screening or confirmatory test due to the following reasons- reducing substances in the lacrimal gland secretions and nasal mucus secretions gives a false positive value, glucose concentration of 5 mg/dl can give a positive value in this test, meningitis can lower the CSF glucose value and causes a false negative result. Hence, this test is not used for confirmation of CSF.

Beta- 2 transferrin test -It is produced by neuraminidase activity in the central nervous system. Apart from CSF, t is also present in the perilymph and aqueous humor. Only a few drops(0.5 ml) of CSF rhinorrhoea is obtained for the test , which may be difficult in cases of intermittent CSF leaks. Only about 10 µL is

needed for the electrophoresis which takes about 120-150 minutes. It is stable at room temperature for 4 hrs and immediate refrigeration following collection is needed. It should not be frozen. It is a confirmatory test for CSF analysis .It has a100 % sensitivity and 95% specificity rate. Patient's serum sample is taken in parallel to avoid false positive[6] results in chronic alcoholism due to chronic liver disease, inborn errors of metabolism, genetic variation of transferrin, neuropsychiatric disorders and rectal carcinoma. The only drawback is its cost and sparsity of centers undertaking this test.

Beta – trace proteins- It is a prostaglandin-D synthase produced by the epithelial cells of choroid plexus, oligodendrocytes and arachnoid cells of central nervous system with CSF concentration 35 fold greater than in plasma. It sis also present in human testes and heart. Minimal sample of 200 µl is enough for the test which takes about 20 mins. Immunoelectrophoresis for its detection gives a sensitivity of 91% and specificity of 100%.

RADIOLOGICAL INVESTIGATIONS

Computed Tomography (CT) scanning- High resolution CT scanning is the radiological investigation of choice to identify the

site of bony defect at the skull base that has resulted from an accidental or iatrogenic trauma, developmental abnormality, tumor etc. The CT scanning is done in axial plane with 1mm slice thickness and coronal and saggital reconstructions are made. Axial images are preferred in detecting defects in the posterior wall of frontal sinus and sphenoid sinus[6].

It may reveal an asymmetry in olfactory fossa on both sides which is suspicious of a defect in this area or an air-fluid level and opacification of paranasal sinuses which is suspicious of CSF accumulation in the presence of relevant clinical findings.It is also not necessary ,that a patient with a skull base defect on CT scan must have an active CSF rhinorrhoea.However, it is mandatory to correlate clinically as infective and allergic rhinitis may have similar CT findings.

A deviated crista galli is a radiological sign in primary CSF leaks in cases of congenital bony defects.A 3- D reconstruction ca help in determining the depth of defect in cases of congenital leaks[18].

CT- cisternography[16]- It is an effective tool in identifying the site of leak.It is done by intrathecal administration of contrast

material like metrizimide or the newer non-ionic low osmolar agent, iohexol (used in our institution) of about 3-10 ml by lumbar puncture and the patient is made to lie in the Trendelenberg's position. An increase in Hounsfield units of 50% or more comparing the pre- contrast and post- contrast scans is diagnostic of CSF leak.Its sensitivity in active leaks is 92% while in inactive leaks is 40%..

Its is most useful in frontal and sphenoid sinus leaks as they act as reservoirs.In the region of cribriform plate, the contrast material track down into the nasopharynz and are less detected by imaging.

Disadvantages are the adverse reactions to the contrast agent which may present with mild symptoms like nausea , vomiting, urticarial, or severe life threatening reactions such as vasovagal shock, bronchospasm ,and karyngeal edema .In addition, iodinated contrast agents are nephrotoxic.

MRI[20] and MR cisternography- MRI is not a first line investigation as it doesnot delineate bony defect, it sis costlier and time consuming..It requires injection of conrtrast in many circumstances.

T_2 - MRI is preferred by particularly in cases with encephaloceles to delineate the contents and vascularity of the sac .It is also done in cases with other intracranial lesions leadind to raised ICP.

MR cisternography involves T_2 –weighted fast spin – echo sequence with fat suppression and subtraction of the adjacent background tissue signal for better identification of the CSF leak. It avoids any intrathecal administration of contrast agents.It has a sensitivity of 87% and accuracy of 89% .

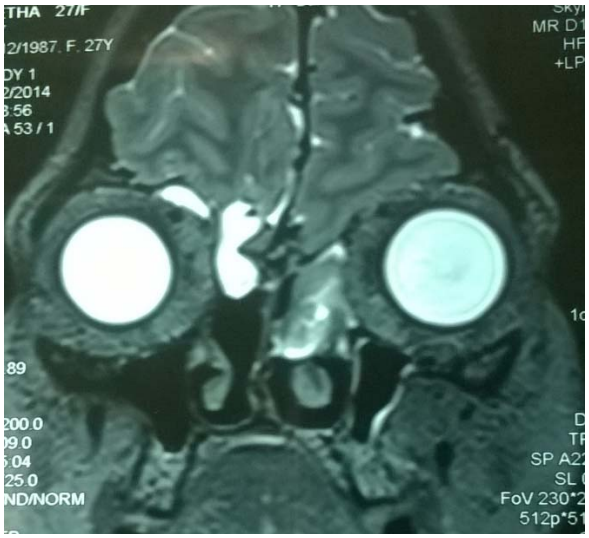


Fig 16: MR- cisternography showing encephalocele on left and CSF leak on right.

RADIOACTIVE CISTERNOGRAM/ NUCLEAR MEDICINE STUDIES[21]

This technique uses intrathecal administration of a radionuclide tacer such as technetium 99m- labelled DTPA

(diethylenetriaminepentaacetic acid , short half-life of 6 hours) and imaging done after 2-4 hrs..Accumulation of the radiotracer in nasal cavity or nasopharynx is indicative of CSF leak.

Its utility is increases by placing nasal pledgets in the region of anterior cribriform plate, the middle meatus, and the sphenoethmoidal recess. To identify the sit eof leak. The pledgets are removed after several hours, and the amount radioactivity in the pledgets is measured and compared with that of serum.The test is positive for CSF leak if pledget –to-serum activity is 1.5-3.0 :1.

For intermittent CSF leak, Indium -111 DTPA is avalalble with longer half- life of 2.8 days. Other tracers include, radioactive iodine -131, ytterbium-169,radioactive serum albumin(RISA) ,technetium =99m human serum albumin and 99mTc pertechnetate

Disadvantages of this study includes,

1) Localisation of the defect is not precise

2) Isotope gets absorbed into the circulatory system contaminating the extracranial tissues

3) Dislodgment of pledgets

4) Radioactivity should be high to determine a leak.

5) False positive rate is high about 33%

ENDOSCOPIC EVALUATION OF CSF LEAKS

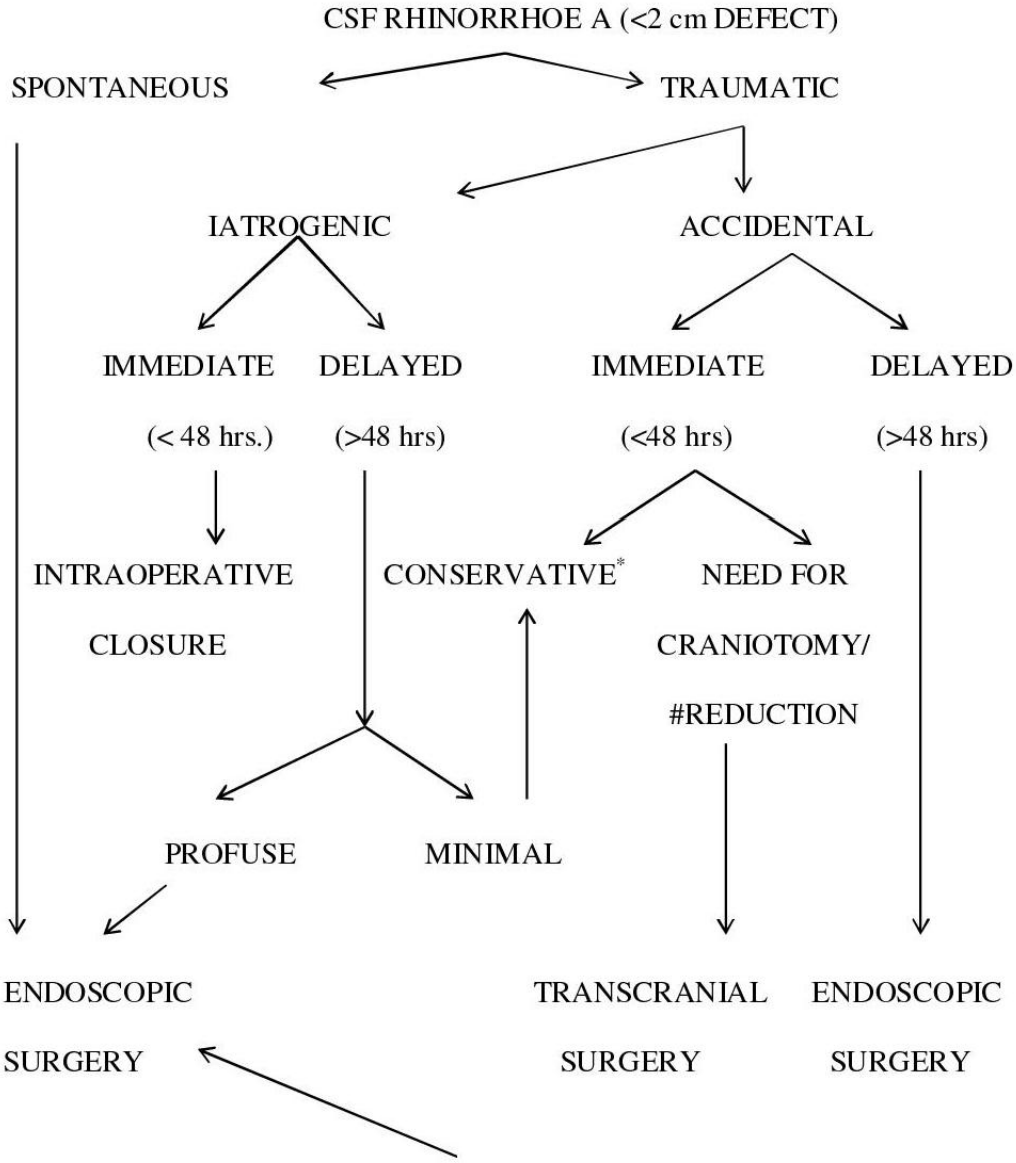
Diagnostic nasal endoscopy- done under strict aseptic precautions using 0^0 Hopkins rod lens endoscope.It detects site of leak when it is active.

Intrathecal fluorescein[22] can be injected intrathecally in a concentration of 0.2 ml of 5% fluorescein diluted in 10 ml of patient's own CSF and reinjected at the rate of 1ml/min.. Nasal endoscopy is done after 30 minutes . This can be done intraoperatively to identify the precise site of leak.

Flourescein is observable using standard xenon light source. Observation under green light with a blue light filter will enhance the visualization of fluorescein.The absobtion range is between 440 – 490 nm..It has to be noted that the US Food and Drug Administration has not approved its use in diagnosis of CSF rhinorrhoea.

MANAGEMENT-

OVERVIEW OF TREATMENT



*FAILURE OF CONSERVATIVE THERAPY >10 DAYS

MEDICAL MANAGEMENT

Includes,

- Head-end elevation of 30^0.
- Avoid sneezing , coughing, straining which may increase ICP
- Stool softeners
- Antibiotics- Most common pathogen in ascending infection onto the exposed intracranium is pneumococcus. Two recent meta-analyses of patients presenting with nonsurgical traumatic CSF leaks revealed no difference in the rates of ascending meningitis in patients treated with prophylactic antibiotics compared with patients treated with conservative measures alone. The use of prophylactic antibiotics can cause meningitis by more virulent and resistant organisms , thus posing difficulty in treatment and affecting the healing post-operatively .
- Although ,prophylactive antibiotic in traumatic CSF leaks have shown to reduce the incidence of meningitis from 61% to 34% in post-traumatic leaks[25] .

- Diuretics- Acetazolamide is a nonbacteriocidal sulfonamide that is used primarily as a diuretic. It is a carbonic anhydrase inhibitor which reversibly inhibits conversion of water and CO2 to bicarbonate and hydrogen ions. The relative deficiency of hydrogen ions within epithelial cells results in decreased Na/K ATPase activity, which results in decreased efflux of water into the CSF. Ultimately, this reduces the volume of CSF. The side effects of acetazolamide include weight loss, diarrhea, nausea, metabolic acidosis, polyuria, and paresthesias. Metabolic profiles should be monitored regularly to monitor its effect on serum electrolytes.

- Lumbar drain[16]- While placing a lumbar rain head end elevation more than 10^0 or sitting up is better avoided to prevent excessive drainage. It is adjusted at the shoulder level to allow a drain rate of 5-10 ml/hr and can be kept insitu for 3-5 days. The drain rate needs to be monitored .If the drain is positioned too low, brainstem herniation can occur due to excessive drainage and placement of the drain at a higher level can cause pnuemocephalous due to siphon effect.

SURGICAL MANAGEMENT

INDICATIONS

Traumatic CSF leaks has a higher resolution rate with medical management. Hence, a waiting period of 7-10 days is allowed with bed rest, head end elevation of $30^{0,}$ stool softeners, lumbar drain. About 70 % close with conservative management.[16] wiyhin 1 week. .If the leak persists more than 10 days[17] of medical therapy, surgery closure of the dural defect must be considered. In cases of delayed post-traumatic CSF leaks, early surgical intervention is favoured.

Iatrogenic injuries warrant immediate intraoperative repair. If it occurs in postoperative period, minimal leaks can be managed conservatively with a spinal tap or lumbar drain , while profuse CSF leaks needs to be surgically addressed.

Primary spontaneous CSF leaks , as it has a very low resolution rate with medical therapy, need to be closed surgically at the earliest. A retrospective study from Mayo clinic showed all the three spontaneous leaks treated medically recurred during a follow up period of 2 years[17].In cases of secondary spontaneous leaks, the associated intracranial pathology need to be surgically addressed ,

such as shunt placement , resection of tumor and reduction of encephalocele , along with dural repair.

In literature even defects of size 3 cm have been endoscopically repaired successfully.

CONTRAINDICATION FOR ENDOSCOPIC SURGICAL REPAIR- Endoscopic repair is contraindicated in patients with poor general condition , who are medically not fit for general anaesthesia, multiple ,comminuted fracture sites, broadly deformed skull base, large bilateral defects , high pressure CSF leaks requiring CSF diversion procedures need intracranial intervention.

SURGICAL TECHNIQUE

INTRACRANIAL APPROACH

In 1926, Dandy described the first intracranial repair through frontal craniotomy. This technique was commonly practiced until 20th century before the advent of endoscope. It had the advantage of direct visualization o fthe cerebral cortex and ability to seal the leak in presence of raised ICP along with shunting procedures and seal larger skull base defects. Cases requiring neurosurgical repair by craniotomy are those with multiple, comminuted fractures, broadly attenuated or badly deformed skull base, tumors with intracranial extension.

Frontal craniotomy, middle and posterior fossa craniotomies were performed to approach the site of leak. Advantage of this method is that , even when preoperative localization of defect fails, blind repair were successful and use of vascularized pericranial flap for repair[29]. Patients requiring osteoplastic flap reconstruction and cranialisation of sinuses required intracranial approach. But, there were higher morbidity , permanent anosmia ,brain retractions with cognitive dysfunction, seizure, prolonged hospital stay, higher incidence of meningitis as compared to endoscopic repair. The overall success rate of this technique is about 60-80%

EXTRACRANIAL APPROACH-

In 1948, Dohlman performed the first extracranial repair for CSF leak through a naso-orbital incision. It is divided into external approach and endoscopic approach.

EXTERNAL APPROACH[30]

Defects in the posterior table of frontal sinus , larger defects 2 cm above the floor and lateral to lamina papyracea can be approached through an external frontoethmoidal incision .The osteoperiosteal flap elevated creates better exposure and create an cosmetically better result .

It can be approached through transorbital or the transnasal approach for access to the ethmoidal and sphenoidal sinus. It includes external ethmoidectommy, transethmoidal sphenoidotomy, trans septal sphenoidaotomy and transantral approach to skull base.

ENDOSCOPIC REPAIR[16]

The first endoscopic repair of CSF leak by Wigand in 1981 has revolutionized the strategies of skull base repair with reported success rate reaching greater than 90% with low morbidity.

GENERAL PRINCIPLES

The principle of skull base-base defect and leak closure is to positively identify the leak site, meticulous preparation of the recipient bed, and accurate placement of the appropriate bed..

Under general anaesthesia, with 15^0 head end elevation , using 0^0 and 45^0 rod lens endoscope and xenon light source ,nasal endoscopy done and the mucosa adequately decongested with topical 0.05% oxymetazoline or 4% cocaine solution .Infilteration of lateral nasal wall with 1% lidocaine with 1:1,00,000 epinephrine promotes vasoconstriction and hemostasis during surgery.

The endoscopic approach gives a better field of vision, enhanced illumination and angled visualization . Unlike in open

techniques, mucosa can be cleaned off the bed without increasing the size of the defec, and the graft can be places accurately.

For better localizing the leak site and to avoid scarring induced secondary sinusitis, a complete ethmoidectomy, maxillary antrostomy is done and when required septal correction, frontal sinusotomy, wide sphenoidotomy, middle- superior turbinectomies may be required for additional exposure. This will also reduce the incidence of iatrogenic mucoceles and collateral damage .

In the initial stages of exposure, grafting has to be planned. Harvesting free septal flaps, septal cartilage, preserving posteriorly based pedicled nasoseptal flaps, preserving the middle turbinate for rotation onto the leak site are all considered .

For positive identification of the leak site, a Valsalva maneuver with the help of anaesthetist or intrathecal fluorescein injection can be done in difficult cases.

EXPOSURE OF SPHENOID AND FRONTAL DEFECTS

Sphenoidal defect can be addressed by transethmoidal approach with wide sphenoidotomy or the midline transseptal approach. Mucosa around the defect has to be carefully removed particularly in regions of the optic canal and the carotid canal can

be dehiscent. Defects in he lateral recess is difficult to approach though this method and hence, trans-pterygoid approach is performed. Wide maxillary antrostomy is done and posterior wall of maxillary sinus removed . The pterygopalatine fossa is entered. Maxillary nerve, vidian nerve and the sphenopalatine ganglion is dissected free and preserved. The anterior wall of sphenoid sinus pneumatizing the pterygoid plates is drilled to enter the lateral recess .

Defects at the isthmus of frontal outflow tract is better address enscopically. This is the region where the skull base transitions from horizontal orientation of the ethmoidal roof to a vertical orientation of the posterior table. The frontal defects are addressed unsing Draf 3 (Modified Endoscopic Lothrop) procedure for wide exposure. In a study[28],the success rate on first attempt was 91.9% (34/37), but improved to 97.3% on subsequent endoscopic revision of frontal sinus leak repair.

PREPARATION OF RECEPIENT BED-

Several millimeters of mucosa surrounding the bony defect is remove to prevent its mucus secretions from separating the graft from the bed and to expose the underlying bone.A diamond burr or a curette can be used to abrade the defect to stimulate osteogenesis.

Encephalocele donot have its function , hence cauterized using a bipolar cautery. Suction monopolar cautery can be used, but with caution as it has higher lateral thermal injury. Hence it is avoided in the region of lamina papyracea and optic nerve. Complete hemostasis has to be obtained at this stage by cauterizing the stalk to avoid potential intracranial hemorrhage. Conventional cautery produces 300^0 C of heat while, saline cautery prevents it to raise above 100^0 hence reducing the lateral damage[27].

The dura is gently elevated from the bone around the defect using otologic elevator to create a plane between the two for the graft to rest in the epidural space.

GRAFTS IN CSF LEAK REPAIR

Free graft guides wound dural healing and acts as a scafflold. They adhere to the bone in 1 week and replaced by fibrous connective tissue at 3 weeks post-operatively. Pedicled grafts has the advantage of vascular supply and long term support for the leak site.

- Fat- harvested from thigh, abdomen, ear lobule . It is an autologous graft. It is an excellent material for sinus obliteration following grafting as it is completely absorbed

when it looses its vascularization. Hence, it doesnot interfere with the function of sinuses following grafting.

- Tensor fascia lata – Unlike in otological grafting, thicker the graft better the result. The size of the graft should be appropriately designed such that , 5 mm of graft lies around the defect area extradurally in the plane between the dura and the bone as underlay technique. It can either be placed as a first layer or over a fat graft.

- Pedicled nasoseptal flap (Hadad flap) - It is based on posterior septal branch of sphenopalatine artery which is used in augmentation in larger defects, frontal sinus defects to additionally line the exposed bone. The only disadvantage is its interposition during instrumentation intraoperatively. This can be avoided by temporarily lodging the flap in the choana and finally repositioning onto the initial layers of closure.

- Cartilage graft – commonly taken from the septal cartilage obtained during septal correction. Tragal or conchal cartilage can also be harvested.

- Composite graft- It includes bone covered by mucosa obtained from middle turbinate. Burns et al[31] discussed

altering the method of closure depending on the size of the defect in the cribriform plate and fovea ethmoidalis. They advocated a free mucosal graft for defects smaller than 0.5 cm. For defects larger than 0.5 cm, they advocated a composite graft with rigid support from a turbinate bone or septal cartilage.

- Bone graft from the septal bone.
- Free muscle graft

TECHNIQUES FOR GRAFTING

OVERLAY- It is used if there was a risk that nerves or vessels might be damaged when raising the dura from the surrounding bone or when inserting the graft, or if an inlay technique was not technically possible. The graft was placed over the dural lesion and over exposed bony margins that had been denuded of mucosa. The graft is then supported in place with layers of gelfoam , surgical, fibrin glue.

UNDERLAY- The dura is separated from the edge of the bony defect to expose an adequate buttress for stable graft insertion. The graft is designed in such a way that it can be pushed

a few millimeters between the bone and the raised intact dura on all sides of the defect.

Bath-plug technique- Wormald and McDonough17 in 1997 presented the “bath plug” technique, which consisted of introducing a fat plug with a secured Vicryl suture into the intradural space.

DURAL SUTURING

For water-tight dural closure, fascial graft is sutured with the dura using 5-0 nylon. It is used for larger defects and in patients with raised ICP.

TECHNIQUE OF LAYERED CLOSURE

To provide adequate support to prevent recurrence, layered closure is widely used. First layer involves fat/fascia, second layer of fascia , third layer of surgicel , followed by gelfoam and tissue sealant. These layers can further be augmented using pedicled and composite grafts discussed earlier.

SANDWICH GRAFTING TECHNIQUE

Two layers of tensor fascia lata is placed in underlay and onlay technique with a layer of cartilage or bone graft inbetween.

FIBRIN SEALANT (TISSEEL/TISSUCOL)- It has two components – a freeze dried concentrate of fibrinogen , factor XIII ,

fibronectin (sealant) and freeze dried thrombin (catalase) First component is reconstituted with aprotinin solution that inhibits tissue fibrinolysis. The second component in 500 IU is dissolved with calcium chloride. It has to be prepared just before its use. It has a hemostatic property by activating the final stages of clotting mechanism as well as wound healing properties.

According to Hassan et al , fibrin glue was used with or without packing in 52% (119/227) of the cases. The success rate was high. However, analysis of his data showed that the results using any of these techniques was not significantly different from each other.

Surgicel- It is made up of oxidized cellulose polymer (polyanhydroglucuronic acid). It has hemostatic and bactericidal property. It gets absorbed over 3-4 weeks.

Gelfoam- A porous hemostat made of purified porcine skin. It is completely absorbed in 4-6 weeks.

POST-OPERATIVE MANAGEMENT AND FOLLOW-UP

Medical therapy , as discussed earlier , was continued for 2-3 weeks with strict bed rest and head end elevation . Lumbar drain when placed is removed after 5-7 days. Merocel pack is placed after 5 days under the cover of antibiotics. Post- operative

endoscopy done on day 5 following removal of merocel pack and 2 weeks later. Patient id followed up once a month for first three months and then every 6 months during the study period.

Fig16: Post- operative nasal endoscopy showing the repaired CSF leak site.

MATERIALS AND METHODS

The study included 22 patients with CSF rhinorrhoea arising from the anterior and midde cranial fossa not subsiding with medical management .It is a prospective study done during the period of 2013 to 2015 in, The Upgraded Institute of Otorhinolaryngology ,Madras Medical College,Chennai.

All the patients were evaluated for CSF rhinorrhoea using a battery of tests which involves the clinical examination for the reservoir sign, biochemical and microbiological analysis of the fluid , radiological investigations and diagnostic nasal endoscopy to assess the site of leak.

All patients were treated in a multidiscilplinary approach. We worked in co-ordination with the neurosurgery department of our Institute in evaluating and treating patients with traumatic history. Some of them needed a neurosurgical intervention for head injury and were managed medically for CSF rhinorrhoea . Patients who didnot respond to the medical management and had a size of defect less than 2 cm were taken up for our study.

In cases of spontaneous CSF rhinorrhoea, we worked in co-ordination with the neurologist to rule out intracranial causes for a raised CSF pressure and all patients were evaluated by ophthalmologist to rule out benign intracranial hypertension as a cause of CSF rhinorrhoea.

This multidisciplinary approach guided us to decide upon further evaluation of a raised intracranial pressure if present and, to plan for the placement of lumbar drain .We evaluated the demographic data, CSF leak site and size, etiology, complications, surgical closure techniques, complications of surgery and recurrences and its management.

WORKING STRATEGY

Patients were evaluated, diagnosed and classified using the following methods,

- History
- Clinical examination
- Rigid nasal endoscopy
- Biochemical and microbiological analysis of CSF
- CT PNS – axial and coronal cuts

- CT – cisternogram / MR cisternogram

Following the investigations the leak site was classified into

- Cribriform plate
- Fovea ethmoidalis
- Sphenoid sinus
- Frontal sinus

Endoscopic closure of the leak site was done as described earlier (refer to surgical management) and the results were analysed.

RESULTS

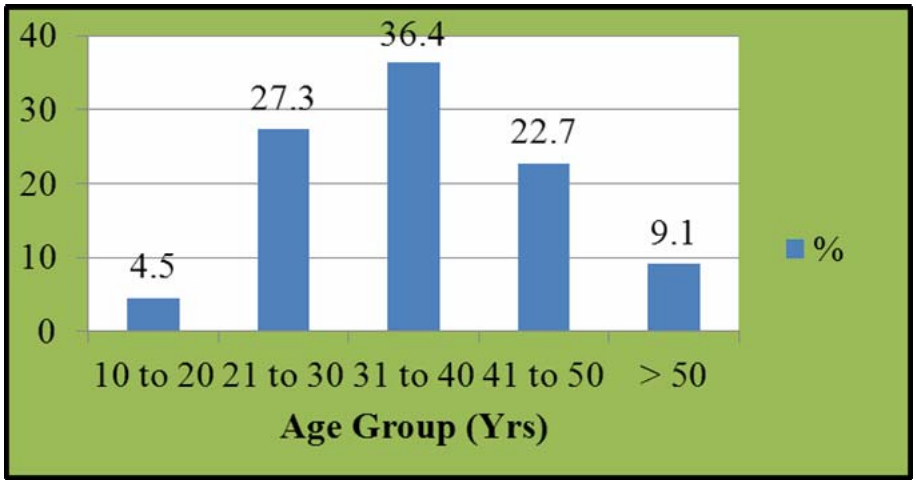
1. AGE GROUP WISE DISTRIBUTION

Table-1: Percentage distribution of study participants by age group

Age Group (Years)	Number	Percentage
10 - 20	1	4.5
21 – 30	6	27.3
31 – 40	8	36.4
41 – 50	5	22.7
More than 50	2	9.1
Total	22	100

Among the study patients, Majority (36.4%) were in 31 – 40 years age group, followed by 21 – 30 years age group (27.3%) and 41 – 50 years (22.7%). The median age of the patients was 34.5 years [Mean age – 36.14 $\pm$ 11.53 years]

Fig-1: Percentage distribution of study participants by age group



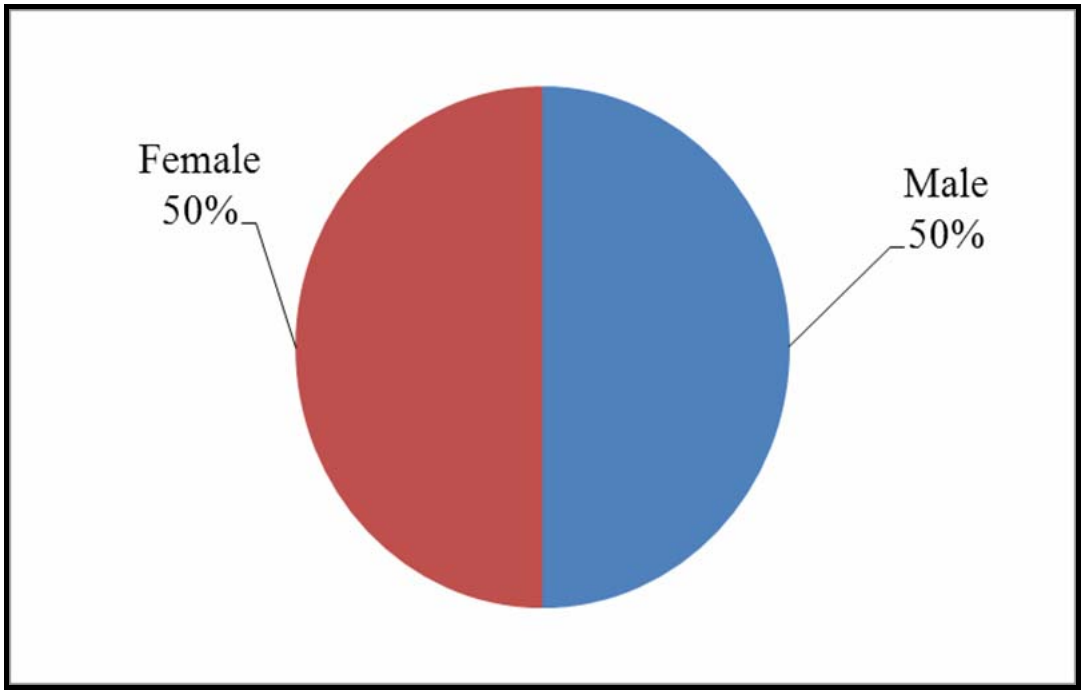
2. SEX DISTRIBUTION

Table-2: Percentage distribution of study participants by sex

Sex	Number	Percentage
Male	11	50
Female	11	50
Total	22	100

Regarding sex distribution of participants, the males and females were found to be equal.

Fig-2: Percentage distribution of study participants by sex



3. PRESENCE OF CO-MORBIDITIES

Table-3: Percentage distribution of study participants by co-morbidities

Co-morbidity	number	percentage
Hypertension	1	4.5
Diabetes mellitus	1	4.5
Meningitis	3	13.6
Neurofibromatosis- 1	1	4.5
Nil	16	72.7
Total	22	100

Of the 22 patients, 3 (13.6 %) presented with meningitis . Two of them had traumatic CSF leak.

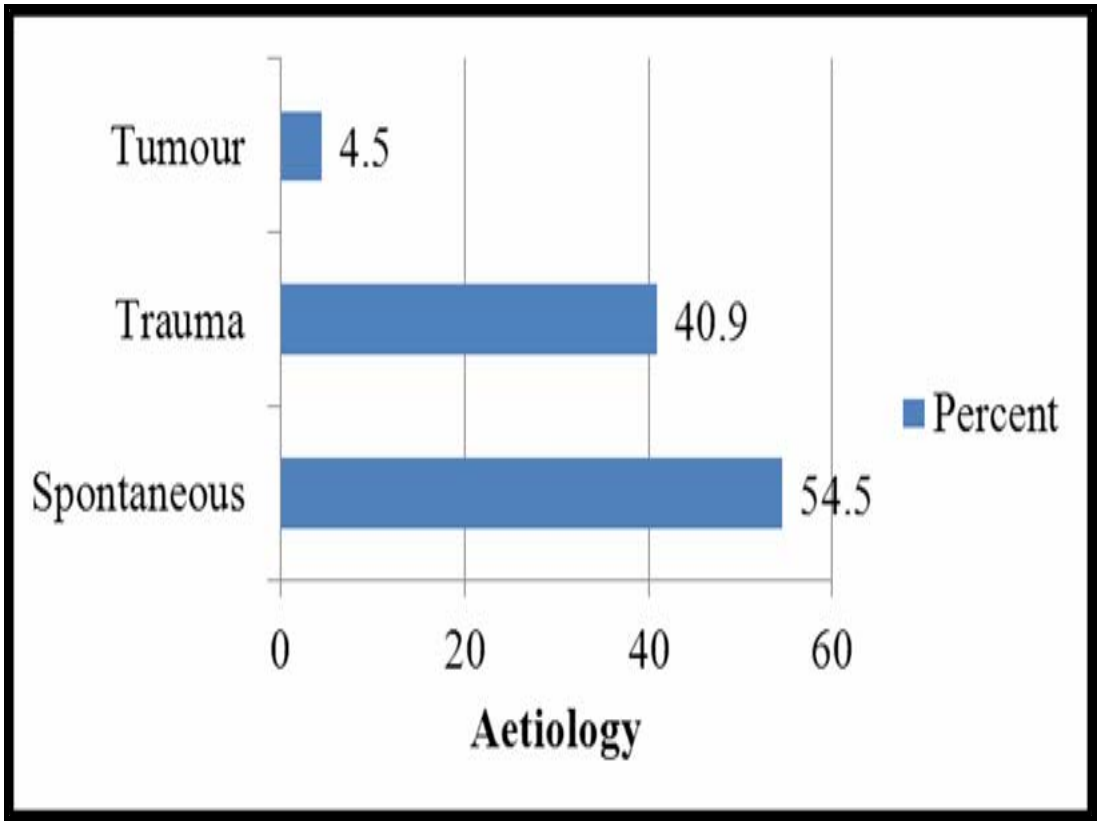
4. ETIOLOGY OF CSF RHINORRHOEA

Table-4: Percentage distribution of study participants by etiology of CSF Rhinorrhoea

Aetiology	Number	Percent
Spontaneous	12	54.5
Trauma	9	40.9
Tumour	1	4.5
Total	22	100.0

Among the study subjects, 54.5% had spontaneous aetiology of CSF rhinorrhoea ,and trauma was reported as cause in 40.9% subjects. One patient (4.5%) had tumour etiology.

Fig-3: Percentage distribution of study participants by aetiology of CSF Rhinorrhoea



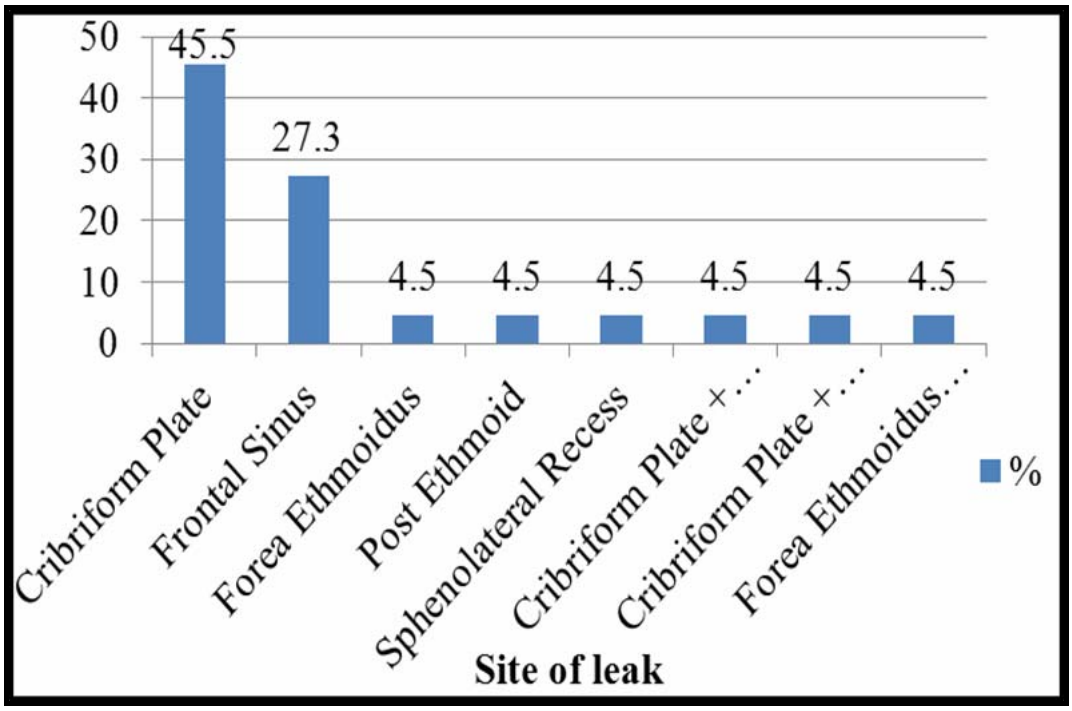
5. SITE OF LEAK

Table-5: Percentage distribution of patients by site of leak

Site of leak	Number	Percent
Cribriform Plate	10	45.5
Frontal Sinus	6	27.3
Fovea Ethmoidus	2	9
Lateral Recess of sphenoid	1	4.5
Cribriform Plate & Fovea Ethmoidalis	1	4.5
Cribriform Plate & Frontal Sinus	1	4.5
Fovea Ethmoidalis & Sphenoidal Sinus	1	4.5
Total	22	100.0

CSF leak was seen commonly, through Cribriform Plate (45.5%) followed by Frontal Sinus (27.3%) and rest through Fovea Ethmoidalis, Posterior Ethmoid, lateral recess of sphenoid and combination of those sites.

Fig-4: Percentage distribution of patients by site of leak



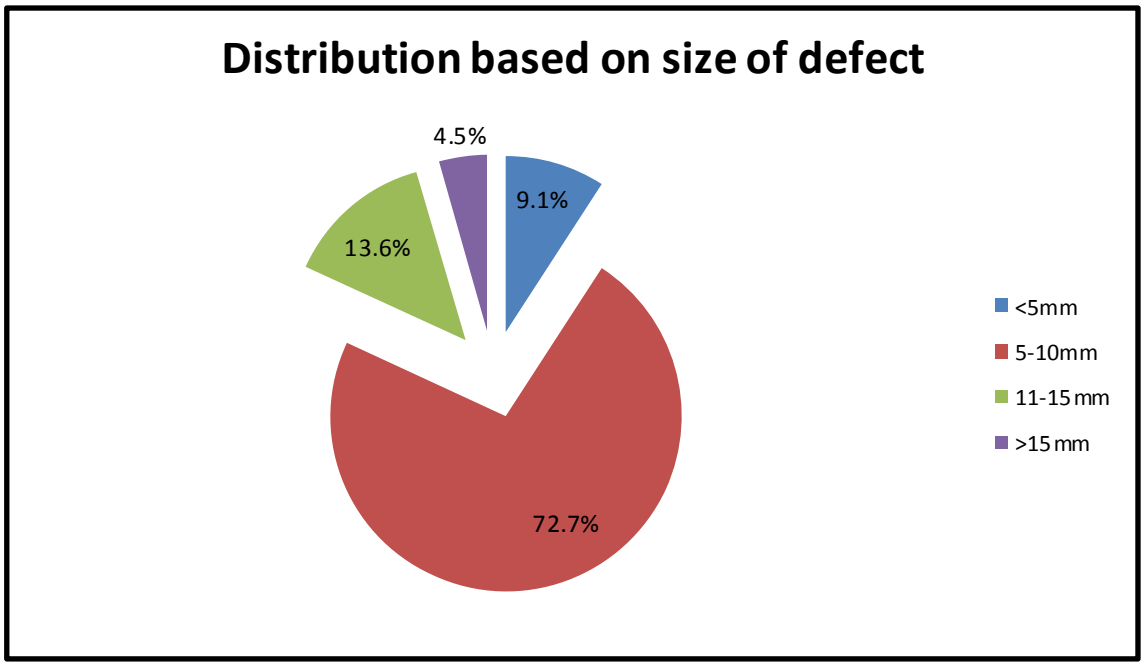
6. SIZE OF LEAK

Table-6: Percentage distribution of patients by size of leak

Size (mm)	Number	Percent
Less than 5	2	9.1
5 – 10	16	72.7
11 – 15	3	13.6
More than 15	1	4.5
Total	22	100.0

Out of 22 subjects, 72.7 % had 5 – 10 mm size of leak and 13.6 % had 11 – 15 mm size of leak. Two had leak size < 5mm and one had > 15mm size.

Fig-5: Percentage distribution of patients by size of leak



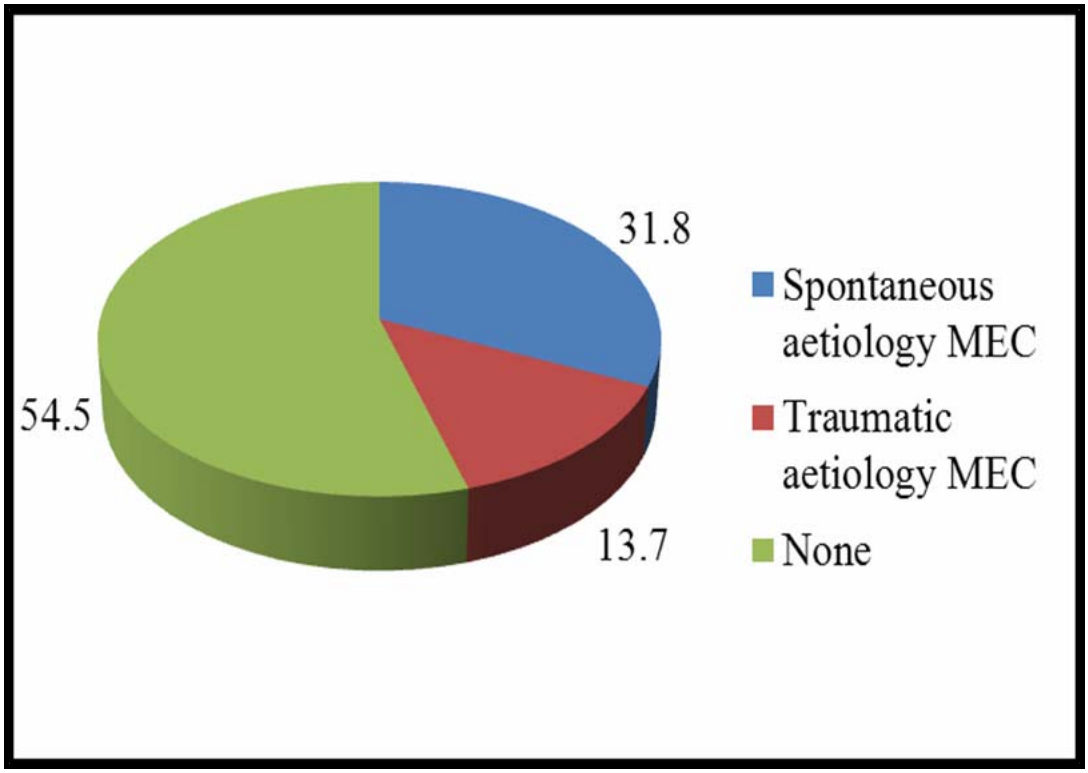
7. ASSOCIATION WITH MENINGOENCEPHALOCELE

Table-7: Percentage distribution of patients by association with Meningoencephalocele

Meningoencephalocele	Number	Percent
Spontaneous aetiology MEC	7	31.8
Traumatic aetiology MEC	3	13.7
None	12	54.5
Total	22	100.0

Meningoencephalocele was seen in 10 subjects (45.5%) and rest 54.5% had no association with meningoencephalocele.

Fig-6: Percentage distribution of patients by association with Meningoencephalocele



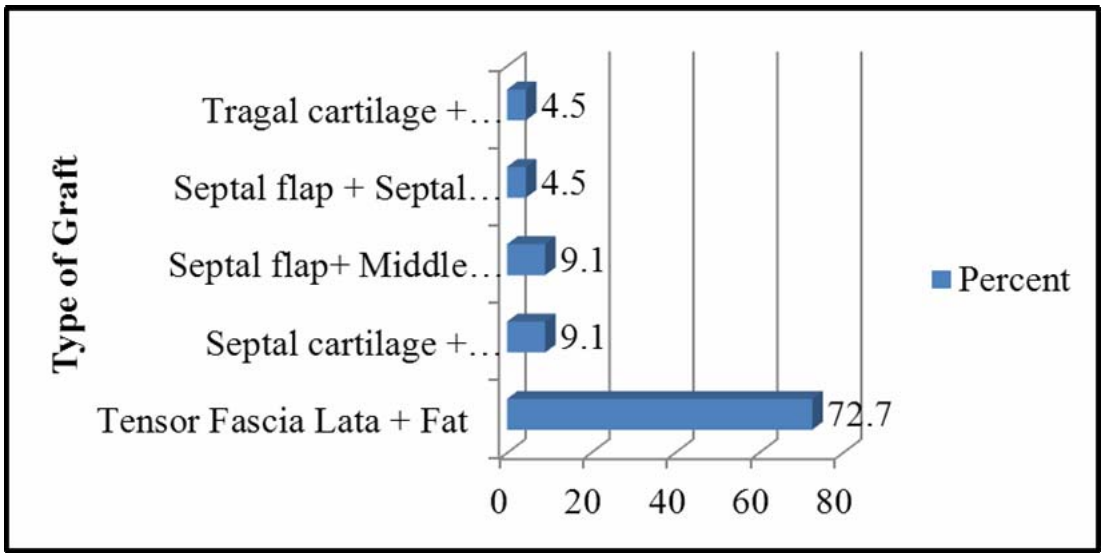
8. TYPE OF GRAFT USED FOR CLOSURE

Table-8: Percentage distribution of patients by type of graft used for closure

Type of graft	Number	Percent
Tensor Fascia Lata & Fat	16	72.7
Septal cartilage & Tensor Fascia Lata	2	9.1
Septal flap & Middle turbinate	2	9.1
Septal flap & Septal cartilage	1	4.5
Tragal cartilage & Middle turbinate	1	4.5
Total	22	100.0

Regarding the type of graft used, majority (72.7 %) of the participants had Tensor fascia lata and fat. Septal cartilage and tensor fascia lata was used in 9.1% cases and septal flap with middle turbinate was used in 9.1% subjects. Rest 9% had tragal cartilage / middle turbinate and septal flap / septal cartilage.

Fig-7: Percentage distribution of patients by type of graft used for closure



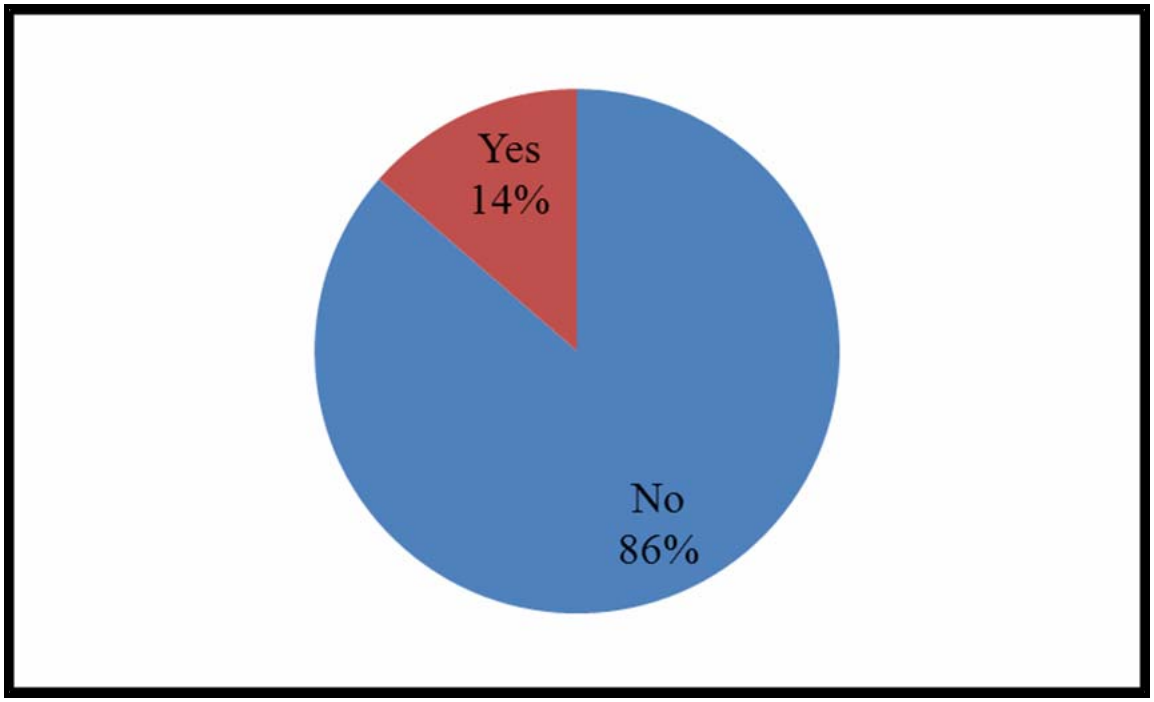
9. LUMBAR DRAIN

Table-9: Percentage distribution of patients by lumbar drain

Lumbar drain	Number	Percent
No	19	86.4
Yes	3	13.6
Total	22	100.0

Lumbar drain was placed in only 13.6% cases and rest (86.4%) had no lumbar drain

Fig-8: Percentage distribution of patients by lumbar drain



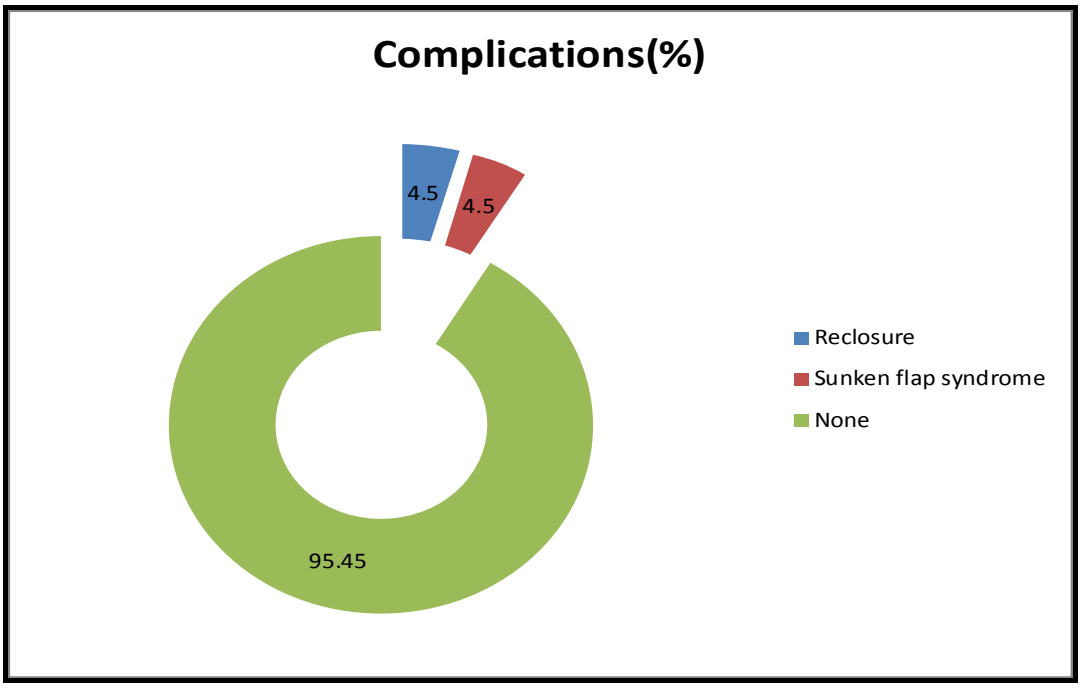
10. COMPLICATIONS AFTER SURGERY

Table-10: Percentage distribution of study participants by complications

Complications	Number	Percent
Reclosure	1	4.5
Sunken flap syndrome	1	4.5
None	20	90.90
Total	22	100.0

Among the study participants, reclosure was done in 1 (4.5%) patients and sunken flap syndrome was seen in one patient (4.5%). CAases without complications was about 90.90% while, successful endoscopic closure with no recurrence was seen in 95.45%

Fig-9: Percentage distribution of study participants by complications



1. AGE GROUPS AND AETIOLOGY

Table-11: Association between age group and aetiology

Age Group (Years)	Aetiology			Total	p value
	Spontaneous	Trauma	Tumour		
10 - 20	0 (0%)	1(100%)	0(0%)	1(100%)	0.009*
21 - 30	1(16.7%)	5(83.3%)	0(0%)	6(100%)	
31 - 40	5(62.5%)	3(37.5%)	0(0%)	8(100%)	
41 - 50	5(100%)	0(0%)	0(0%)	5(100%)	
> 50	1(50%)	0(0%)	1(50%)	2(100%)	

*significant ($p < 0.05$)

Traumatic aetiology was seen mostly among younger age groups (< 40 years) when compared to older age groups, where spontaneous aetiology predominates. This association was found to be statistically significant ($p < 0.05$)

2. AGE GROUP AND SITE OF LEAK

Table-12: Association between age group and site of leak

Age group (Yrs)	Site of leak			Total	p value
	Cribriform Plate	Frontal Sinus	Others		
10 - 20	1(100%)	0(0%)	0(0%)	1(100%)	0.764
21 - 30	1(16.7%)	3(50%)	2(33.3%)	6(100%)	
31 - 40	4(50%)	2(25%)	2(25%)	8(100%)	
41 - 50	3(60%)	1(20%)	1(20%)	5(100%)	
51 - 60	1(50%)	0(0%)	1(50%)	2(100%)	

In all age groups, the site of leak is mainly through cribriform plate, except 21 – 30 years where it is through frontal sinus.

3. AGE GROUP AND MENINGOENCEPHALOCELE (MEC)

Table-13: Association between age group and Meningoencephalocele

Age group (Yrs)	MEC		Total	p value
	Yes	No		
10 - 20	1(100%)	0(0%)	1(100%)	0.377
21 - 30	1(16.7%)	5(83.3%)	6(100%)	
31 - 40	5(62.5%)	3(37.5%)	8(100%)	
41 - 50	2(40%)	3(60%)	5(100%)	
51 - 60	1(50%)	1(50%)	2(100%)	

Meningoencephalocele is seen mainly among 31 – 40 years age group, followed by 41 – 50 year age group.

4. AGE GROUP AND COMPLICATIONS SEEN

Table-14: Association between age group and complication

Age group (Yrs)	Complications		Total	p value
	Nil	Yes		
10 - 20	1(100%)	0(0%)	1(100%)	0.193
21 - 30	6(100%)	0(0%)	6(100%)	
31 - 40	5(62.5%)	3(37.5%)	8(100%)	
41 - 50	5(100%)	0(0%)	5(100%)	
51 - 60	2(100%)	0(0%)	2(100%)	

Complications after surgery is seen only in 31 – 40 year age groups. No complications were reported in other age groups.

5. SEX DISTRIBUTION AND AETIOLOGY

Table-15: Association between sex and aetiology

Sex	Aetiology			Total	p value
	Spontaneous	Trauma	Tumour		
Male	1(9.1%)	9(81.8%)	1(9.1%)	11(100%)	0.0001*
Female	11(100%)	0(0%)	0(0%)	11(100%)	

*significant ($p < 0.05$)

Regarding association between sex distribution and aetiology, 81.8% of the males had trauma, whereas all the females had spontaneous aetiology for CSF Rhinorrhoea. This association was found to be highly statistically significant ($p < 0.0001$)

6. SEX DISTRIBUTION AND SITE OF LEAK

Table-16: Association between sex and site of leak

Sex	Site of leak			Total	p value
	Cribriform Plate	Frontal Sinus	Others		
Male	3(27.3%)	5(45.5%)	3(27.3%)	11(100%)	0.118
Female	7(63.6%)	1(9.1%)	3(27.3%)	11(100%)	

Among male patients, majority had (45.5%) had frontal sinus leak and rest had leak from cribriform plate and other sites. In females, cribriform plate leak was predominant in 63.6% cases, while rest had leak from other sites. The association is not statistically significant ($p > 0.05$)

7. SEX DISTRIBUTION AND MENINGOENCEPHALOCELE (MEC)

Table-17: Association between sex and MEC

Sex	MEC		Total	p value
	Yes	No		
Male	4(36.4%)	7(63.6%)	11(100%)	0.392
Female	6(54.5%)	5(45.5%)	11(100%)	

Meningoencephalocele is associated with 60% of females when compared to males.

8. SEX DISTRIBUTION AND COMPLICATIONS AFTER SURGERY

Table-18: Association between sex and complications

Sex	Complications		Total	p value
	Nil	Yes		
Male	10(90.9%)	1(9.1%)	11(100%)	1.000
Female	10(90.9%)	1(9.1%)	11(100%)	

Complications following surgery is seen equally among males and females on between sex and site of leak.

9. AETIOLOGY AND SITE OF LEAK

Table-19: Association between aetiology and site of leak

Aetiology	Site of leak			Total	p value
	Cribriform Plate	Frontal Sinus	Others		
Spontaneous	7(31.8%)	1(4.5%)	4(18.2%)	12(54.5%)	0.127
Trauma	2(9.1%)	5(22.7%)	2(9.1%)	9(40.9%)	
Tumour	1(4.5%)	0(0%)	0(0%)	1(4.5%)	
Total	10(45.5%)	6(27.3%)	6(27.3%)	22(100%)	

Most common site of leak associated with spontaneous aetiology is cribriform plate (31.8%) and frontal sinus leak is seen more with traumatic aetiology (22.7%).

10. AETIOLOGY AND MENINGOENCEPHALOCELE (MEC)

Table-20: Association between aetiology and MEC

Aetiology	**MEC**		**Total**	**p value**
	Yes	**No**		
Spontaneous	7(31.8%)	5(22.7%)	12(54.5%)	0.338
Trauma	3(13.6%)	6(27.3%)	9(40.9%)	
Tumour	0(0%)	1(4.5%)	1(4.5%)	
Total	10(45.5%)	12(54.5%)	22(100%)	

Spontaneous aetiology is most commonly associated with meningocele (31.8%).

11. AETIOLOGY AND COMPLICATIONS AFTER SURGERY

Table-21: Association between aetiology and complications

Aetiology	Complications		Total	p value
	Nil	Yes		
Spontaneous	11(91.7%)	1(8.3%)	12(100%)	0.926
Trauma	8(88.9%)	1(11.1%)	9(100%)	
Tumour	1(100%)	0(0%)	1(100%)	
Total	20(90.9%)	2(9.1%)	22(100%)	

Complications after surgery is seen equally among spontaneous and traumatic aetiology. Of this recurrence was encountered in spontaneous leak.

12.BODY MASS INDEX (BMI):

Table-21: Percentage distribution of study participants by BMI

BMI category	Number	Percent
< 18.5	1	4.5
18.5 – 24.9	17	77.3
> 25	4	18.2
Total	22	100.0

Majority, 17(77.3%) of the study participants had normal BMI [18.5 – 24.9], while 18.2% are overweight [BMI > 25]. The median BMI value is 22.

13.BMI AND AETIOLOGY:

Table-22: Association between BMI and aetiology

BMI category	Aetiology			Total	p value
	Spontaneous	Trauma	Tumour		
< 18.5	0 (0%)	1(100%)	0(0%)	1(100%)	0.183
18.5 – 24.9	10(58.8%)	7(41.2%)	0(0%)	17(100%)	
> 25	2(50%)	1(25%)	1(25%)	4(100%)	
Total	12(54.5%)	9(40.9%)	1(4.5%)	22(100%)	

Association of BMI and etiology of CSF leak is statistically insignificant.

DISCUSSION

In the above prospective study conducted at the Upgraded Institute of Otorhinolaryngology, Rajiv Gandhi Government General Hospital , 22 patients who were treated through surgical approach after a failed medical therapy were included .The socio-demographic and clinical data were studied and analysed to determine the common sites of CSF leak presenting under the various etiology.

Further, this study also analyses the various methods of the defect closure elucidating our experience in managing the CSF leak intraoperatively and post operatively, and the efficacy of endoscopic closure of these skull base defect.

Among the study group, majority of patients belong to the age group of 31-40 yrs. Followed by 21-30 yrs. Our study included an equal proportions of male and female patients. Of the total 22 patients, 12 (54.5%) presented with spontaneous CSF rhinorrhoea and 9 patients (40.9 %) presented with post traumatic CSF rhinorrhoea.Based on the age distribution of the cases, traumatic CSF rhinorrhoea is seen more commonly in younger age groups

<40 yrs while spontaneous CSF leak is seen in older age groups which is **statistically significant.**

Also, all except one spontaneous leak presented in female patients. This is an important observation relating to the social cause , where males are more commonly involved in road traffic accidents and other causes of accidental injury and related trauma. 81.8% of male patients had traumatic CSF leak which gives a p vale for association of etiology and sex distribution , a **statistically significant** value of p=0.0001.This is consistent with the study by Beckhardt et al in 1991 . He observed spontaneous leaks to be more common in females (male:female ratio =1:2)[34]

Females are more prone for spontaneous leaks ,of which we have not encountered any cases of idiopathic intracranial hypertension as a reason of the leak. There were no known causes of CSF leak in these patients.

Of the total spontaneous leaks, one female patient had a history of previous three endoscopic closure presenting with recurrence to us. Following all investigations, intracranial hypertension was ruled out and meningoencephalocele was identified to be the only conclusive finding for a non-healing dura

and recurrence. Hence, she was taken up for revision endoscopic surgery with adjunctive medical therapy , without placement of lumbar drain. The patient was followed up for 1yr and didnot have any recurrence.

Regarding the sex distribution across the site of leak, 45.5% of male patients had leak from frontal sinus and 63.6% of females had leak from the cribriform area, though this difference was statistically not significant , $p>0.05$. We encountered a case of spontaneous CSF leak in a female patient from the roof of frontal sinus- a rare case of spontaneous CSF leak.

In our study, the most common site of leak associated with spontaneous aetiology is cribriform plate (31.8%) and frontal sinus leak is seen more with traumatic aetiology (22.7%). According to Kennedy[33] site of CSF leaks among 27 patients found to be 17 patients with leak in ethmoids, while 6 patients in the cribriform plate and in the sphenoid in 4 patients.

D.Richard Lindstrom et al. experience in Wisconsin Medical college is given in number of patients as, Cribriform plate -20; Anterior ethmoid-10; Posterior ethmoid- 5; Frontal-6; Sphenoid -15

During our study, we also encountered three cases presenting with two sites of leak. Of them one spontaneous and one traumatic leak had two non-adjacent sites of leak. This is an unusual finding not nuch reported in the literature, especially in the spontaneous variety. The defects were closed independently after ruling out other intracranial pathology. These cases were also followed up and no recurrence was encountered. In every patient who undergoes imaging evaluation for a CSF leak, potential multiplicity of defects has to be borne in mind. Schlosser and Bolger[23] recognized multiple simultaneous skull base defects during surgery in 5 (31.25%) of 16 patients with nasal CSF leaks

Three patients presenting with features of meningeal irritation were initially treated with third generation cephalosporins, T.acetazolamide, and T.phenobarbitone before definitive surgical intervention was made.

Lumbar drain was placed in three traumatic cases were CSF pressure re was needed. These cases had a bony defect of 1 cm or more and the drain was placed for 3-5 days .One of these cases developed sunken flap syndrome or the motor trephine syndrome, wherein which, due to bifrontal craniotomy done previously, atmospheric pressure far exceeded the intracranial pressure ,

causing the forehead skin to retract into the cranium. This lead to slowness of motor activites. Lumbar drain was removed and the dose of T.acetazolamide was reduced. Adequate hydration was provided , keeping in balance the intracranial pressure ,so as to avoid a raised ICP that would precipitate a CSF leak closure failure. Following this treatment , patient's neurological status improved; CSF leak well sealed , and planned for cranial flap reconstruction by the neurosurgical team.

Different methods of endoscopic leak closure were studied of which, the underlay and the sandwich techniques were preferred by the surgeons in larger defects and all these cases were successfully treated. One out of the four cases of overlay technique failed due to difficulty in localizing the defect, which was reclosed using the same technique and was successful. Although the surgeons generally restrict the overlay technique to smaller defects, the success rate of different techniques studied were high.

One out of the 22 patients developed recurrence (success rate of 95.45%), and needed a revision closure within 3 weeks of the previous surgery. Intraoperatively, active CSF leak was not encountered in this patient, which we consider as the cause for the recurrence. The patient was adviced a repeat CT – cisternogram

and the site of leak indentified during the revision surgery and the result was successful immediately, as demonsrated by the Valsalva test done with the help of anaesthetist. Meta-analysis by Hassan et al reported a 90%success rate[24] of endoscopic repair following first attempt , and a 97% success rate following revision surgery. The incidence of major complications such as meningitis, abcess, subdural hematoma were less than 1% each. Hence, it is considered to be less morbid than open surgical techniques.

In a study[35], all patients with spontaneous CSF leaks were overweight (BMI, >25 kg/m2) with an average BMI of 37.8 kg/m2. The average age was 57.03 years, and 72% were female. All our patients with spontaneous had BMI of about 25 or less, except one male patient (BMI=26).

CONCLUSION

We conclude that, in our study, the most common site of leak associated with spontaneous etiology is cribriform plate (31.8%) and frontal sinus leak is seen more with traumatic etiology (22.7%). The method of closure may vary. But, the identification of the leak site and the plane between dura and the bone around the defect is important determinant of the success rather than the choice of material. For most of these cases, fat was used as the first layer of underlay technique which acts as a good seal. Additional procedures such as pedicled nasoseptal flap, rotation of middle turbinate, septal cartilage augmentation were used in larger defects of about 6 mm or more.

The efficacy of endoscopic CSF leak closure in our study was 95.45%.

PROFORMA

Name: Date:

Age/sex: IP/OP no:

Occupation:

Chief complaints:

H/O presenting illness:

	Complaints	Yes	No	Duration
1	H/O watery nasal discharge			
2	H/O increased nasal discharge during straining			
3	H/O nasal obstruction			
4	H/O epistaxis			
5	H/O anosmia			
6	H/O fever			
7	H/O headache			
8	H/O vomitting			
9	H/O seizures			
10	H/O altered sensorium			
11	H/O blurring of vision			
12	H/O trauma			

Past History: Diabetes mellitus/ Hypertension/ epilepsy/ asthma/jaundice.

Treatment history: Previous nasal surgery/ H/O similar episode in the past with surgical or medical treatment for CSF leak / H/O any neurosurgical procedure/chronic medications.

Personal history: Diet/appetite/smoking/alcohol/bladder and bowel habits.

Family history: married / unmarried/ similar illness in any other family members.

GENERAL EXAMINATION

Conciousness: Height:

Orientation: Weight:

Febreile/afebrile: BMI:

Built:

Pallor/icterus/cyanosis/clubbing/lymphadenopathy/pedal edema

Vitals- BP:

Pulse rate:

Respiratory rate:

SYSTEMIC EXAMINATION:

Cardiovascular system:

Respiratory system:

Central nervous system:

Higher functions/cranial nerves/sensory and motor

System/ Pyramidal system/vision/signs of meningeal irritation.

ENT EXAMINATION

Nose:

External contour- deformity/scar

Anterior rhinoscopy- septal deviation/mass/nasal discharge

Posterior rhinoscopy-

Cold spatula test-

Paranasal sinus tenderness-

Reservoir sign-

Handkerchief sign-

Halo sign-

EAR: **right** **left**

Pinna-

External auditory cana-

Tympanic membrane-

Tuning fork test-

Throat:

Oral cavity-

Oropharynx

Indirect laryngoscopy-

INVESTIGATIONS

a)Blood – Hb %TC/DC/ESR/BT/CT/BloodUrea Sugar / Serum

Creatinine

b) Urine – Albumin / Sugar / Microscopy

c) Blood grouping and typing

d) CSF analysis – Glucose estimation

e) CT PNS - Axial and coronal

f) CT/MRI cisternogram.

g) Pre OP Diagnostic Nasal Endoscopy

Diagnosis:

Management:

Medical management-

Surgical management-Site of defect, graft used ,method of closure

Post operative follow-up

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ஆராய்ச்சி தகவல் தாள்

சென்னை ராஜீவ் காந்தி அரசு மருத்துவமனைக்கு வரும் மூளைநீர் மூக்கின் வழி வடிதல் நோயாளிகளின் அறுவை சிகிச்சை பற்றிய ஆய்வு.

மண்டை ஓட்டின் விரிசல் வழியே மூளைதண்டுவடநீர் (cerebro spinal Fluid- CSF) மூக்கின் வழி வெளியேறுதல் CSF RHINORRHOEA எனப்படுகிறது. இதன் வழி கிருமிகள் மூளைக்குப் பரவி மூளைக்காய்ச்சல் ஏற்பட வாய்ப்புள்ளது.

இது, இவ்விரிசலை, உட்குழாய் கொண்டு அடைத்தல் எத்தகு பயனை அளிக்கிறது என்பதைப் பற்றிய ஆராய்ச்சியாகும்.

நீங்கள் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம்.

இந்த ஆராய்ச்சியின் முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி :

INFORMATION TO PARTICIPANTS

Title - **" ENDOSCOPIC REPAIR OF CSF RHINORRHOEA – A REVIEW ON ITS EFFICACY AND SUCCESS RATE".**

Principal investigator :

Co-investigator (if any):

Name of the participant:

Site:

You are invited to take part in this research/study/procedures/tests. The information in this document is meant to help you whether or not to take part. Please feel free to ask if you have any queries or concerns.

What is the purpose of research?

Surgical management of CSF rhinorrhoea can be made more precise and less morbid with endoscopicendonasalapproach . We want to test the efficacy and success rate of a new _____ (drug/intervention/surgery/procedure/lab test) in this disease/condition.

The study design: Prospective study.

Study procedures:

The study involves confirmation of CSF leak and identification of the site of CSF leak for which we will need physicalexamination, biochemical test for CSF, direct endoscopic visualization of defect, CT-PNS – axial and coronal cuts and /or CT / MR – cisternogram. The planned schedule involve visits at _____,_____,_____ and _____ days /weeks after your initial visit. You will be required to visit the hospital _____ number of times during the study.

At each visit the study physician will examine you. Some blood/urine/imaging/clinical examination/other tests will be carried out at each visit if needed. {_____ml of blood will be collected at each visit. Blood collection involves prick with a needle and syringe}.These tests are essential to monitor your condition and assess the safety and efficacy of treatment given to you. In addition if you notice any physical or mental change(s), you must contact the persons listed at the end of the document.

You may have to come to the hospital (study site) for examination and investigations apart from your scheduled visits , if required.

Possible risks to you – If any briefly mention.

Possible benefits to you - If any briefly mention.

Possible benefits to other people

The results of the research may provide benefits to the society in terms of advancement of medical knowledge and/or therapeutic benefit to future patients.

Confidentiality of the information obtained from you

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations and your medical history). By signing the document you will be allowing the research team investigators other study personnel, sponsors, Institutional Ethics Committee and any person or agency required by law like the Drug Controller General of India to view your data if required.

The information of this study if published in scientific journals or presented at scientific meetings will not reveal your identity.

How will your decision not to participate in the study affect you?

Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not loose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from the study at any timeduring the course of the study without giving any reasons. However, it is advisable to talk to the research team prior to stopping the treatment/discontinuing the procedures etc.

Signature of the investigator

Signature of the participant

Date

Date

INFORMED CONSENT FORM

Title of the study - **" ENDOSCOPIC REPAIR OF CSF RHINORRHOEA – A REVIEW ON ITS EFFICACY AND SUCCESS RATE".**

Name of the participant :

Name of the principal / co-investigator :

Name of the institution : Upgraded Institute Of Otorhinolaryngology, Madras Medical College and Rajaji Government General Hospital , Chennai.

I _____ (name of the participant) , have read the information in this form (or it has been read to me). I was free to ask any question and they have been answered. I am over 18 years of age and , exercising my free power of choice, hereby give my consent to be included as a participant in _____" (title of the study).

(!) I have read and understood this consent form and the information provided to me.

(2) I have had the consent document explained to me.

(3) I have been explained about the nature of the study.

(4) I have been explained about my rights and responsibilities by the investigator.

(5) I have informed the investigator of all the treatments I am taking or have been in the past _____ months including my native (alternative) treatments.

(6) I have been advised about the risks associated with my participation in the study.

(7) I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.

(8) I have not participated in any research study within the past _____ months.

(9) I have not donated blood within the past _____ months.

(10) I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in the hospital.

S. No	Name	Age	Sex	BMI	Co-moridities	Previous Closure	Etiology	Duration (months)	Site of Leak	Size (mm)	MEC	Grafting technique
1	Anuradha	42	F	26			S	2	R CP LL	5	mec	Underlay
2	Arun Kumar	33	M	20			T	2	R CP	20	mec	Sandwich
3	Chellamal	64	F	23	HT		S	1	R FE	10	mec	Underlay
4	Elumalai	44	M	25	DM		S	2	R FE, SS (lateral recess)	5,7	mec	Underlay
5	Geetha	27	F	24		Thrice	S	1/2.	L CP	15	mec	Underlay
6	Indrani	34	F	22			S	3	L CP	12		Overlay
7	Kalaiselvi	43	F	24			S	1	R CP LL	10	mec	Underlay
8	Lakshmi	38	F	21	Meningitis, seizures		S	2	L CP	7	mec	Underlay
9	Lovely	27	M	26			T	1	L FE	4		Overlay
10	Manjula	40	F	22			S	6	R CP	5		Overlay
11	Mariadoss	33	M	23			T	18	L FS	10	mec	Underlay by Draf 3
12	Santhosh	34	M	22	Meningitis	Twice	T	1	L FS	7		Overlay by Draf 3
13	Sathyaraj	24	M	22			T	5	L FS	10		Sandwich by Draf 3
14	Selvi	40	F	23			S	6	R CP LL, FE	12	mec	Uderlay
15	Surya	12	M	18	Meningitis		T	24	L CP	6	mec	Underlay
16	Uma	48	F	21	NF 1		S	6	R CP	6		Underlay
17	Valiselvan	55	M	25			Tumor	Intraoperative	L CP	7		Underlay
18	Velmurugan	25	M	22			T	3	P CP, L FS	5,8		Uderlay by Draf 3
19	Ranjith	24	M	21			T	4	R CP	4		Overlay
20	Dillibabu	28	M	23		Once	T	3	R FS	10		Underlay
21	Geetha Prakash	45	F	22			S	4	L CP ML	5		Underlay
22	Valsa Mary	35	F	22			S	4	R SS (lateral recess)	7	mec	Underlay

S. No	Name	Graft used	LD	Reclosure	Complications
1	Anuradha	ear lobule fat+septal cartilage+ hadad flap+SG+TS+SG+GF			
2	Arun Kumar	TFL+septal cartilage+TFL+MT mucosa+SG	Yes		Sunken flap syndrome
3	Chellamal	TFL+TS+SG+thigh fat+TS			
4	Elumalai	FE- TFL+TS; SS-fat+TFL+ septal cartilage+TS+SG			
5	Geetha	TFL+septal catilage+TFL+TS			
6	Indrani	leak site not identified; posterior septal flap +SG+TS		MEC cauterised; Overlay of mucosal graft +SG+TS+remnant MT rotated+TS+SG	
7	Kalaiselvi	tragal cartilage+MT+SG+GF			
8	Lakshmi	thigh fat+TFL+TS+SG			
9	Lovely	fat+TFL+mucosa+SG+TS			
10	Manjula	MT free graft+SG+TS			
11	Mariadoss	Thigh fat+muscle+TFL+SG+TS			
12	Santhosh	fat+hadad flap+TS			
13	Sathyaraj	TFL+Cartilage+TFL+SG+TS	Yes		
14	Selvi	septal cartilage+mucosal flap+SG+GF			
15	Surya	septal cartilage+TS+fat+TS+TFL+muscle			
16	Uma	Fat+TFL+SG+TS+SG+TS			
17	Valiselvan	fat+TFL+TFL+SG+TS			
18	Velmurugan	CP - TFL+SG+TS+SG+hadad flap; FS - TFL+SG+TS+Hadad flap+MT	Yes		
19	Ranjith	Fat+TFL+fat+TS+SG			
20	Dillibabu	TFL+SG+TS+SG			
21	Geetha Prakash	Fat+TFL+TS+SG+TS			
22	Valsa Mary	fat+TFL+SG+TS+SG+TS			

KEY TO MASTER CHART

HT	:	Hypertension
DM	:	Diabetes Mellitus
NF	:	Neurofibromatosis
S	:	Spontaneous
T	:	Traumatic
CP	:	Cribriform Plate
LL	:	Lateral Lamella
SS	:	Sphenoid Sinus
FE	:	Fovea Ethmoidalis
FS	:	Frontal Sinus
MEC	:	Meningoencephalocele
TFL	:	Tensor Fascia Lata
SG	:	Surgicel
TS	:	Tisseel
GF	:	Gelfoam
MT	:	Middle Turbinate

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Hemalatha .K,
PG in Otorhinolaryngology,
Upgraded Institute of Otorhinolaryngology,
Madras Medical College, Chennai-3.

Dear Dr. Hemalatha .K,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Endoscopic Repair of CSF Rhinorrhoea – A review of its Efficacy and Success Rate"** No.01032014

The following members of Ethics Committee were present in the meeting held on 11.03.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Prof. Kalaiselvi, MD Vice-Principal, MMC, Ch-3	-- Member Secretary
3.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
4.	Prof. Bhavani Shankar, M.S. Prof & HOD of General Surgery, MMC, Ch-3.	-- Member
5.	Prof. V. Padmavathi, M.D. I/c Directory of Pathology, MMC, Ch-3.	-- Member
6.	Thiru. S. Govindasamy, BABL	-- Lawyer
7.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

2013/14

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Member Secretary, Ethics Committee

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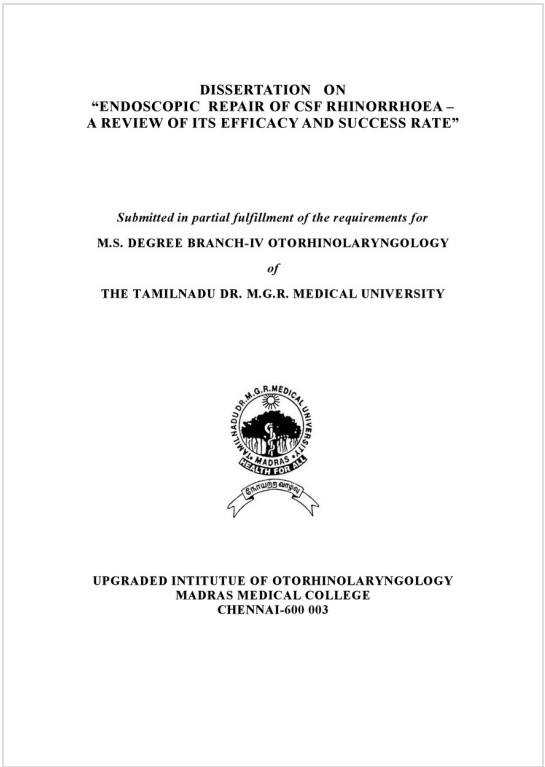

DISSERTATION ON
"ENDOSCOPIC REPAIR OF CSF RHINORRHOEA –
A REVIEW OF ITS EFFICACY AND SUCCESS RATE"

Submitted in partial fulfillment of the requirements for

M.S. DEGREE BRANCH-IV OTORHINOLARYNGOLOGY

of

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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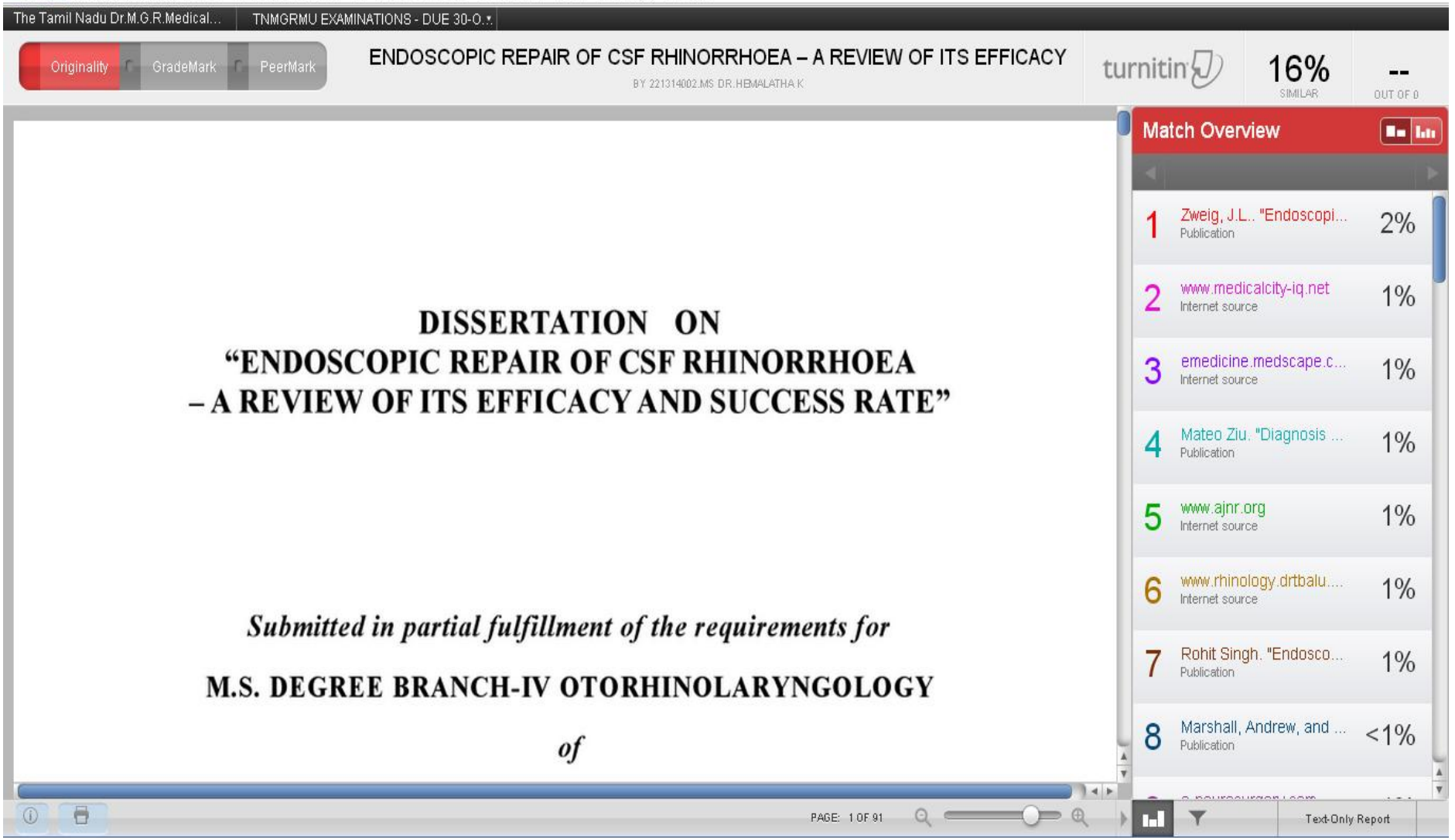
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